



INTERNATIONAL  
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MEDICINE  
EDUCATION  
PROJECT

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*iEmergency Medicine for  
Medical Students and Interns*

1st Edition, Version 1, 2018

2018

**A Free Book For**

# **Emergency Medicine Clerkship**

**Students**

## **Editors**

Arif Alper Cevik

Lit Sin Quek

Abdel Nouredin

Elif Dilek Cakal

# Title Page



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# What is iEM?

**International Emergency Medicine (iEM) Education Project** is an international, non-profit project, endorsed by International Federation for Emergency Medicine (IFEM) and supported by Emergency Medicine professionals from all around the world. Currently, there are **146 contributors from 21 countries** in the project. It aims to promote Emergency Medicine and provide free, reusable educational content for undergraduate medical trainees and educators.

The project focuses mainly on **undergraduate curriculum**, but learners from all levels (medical students, interns, residents) and their educators may benefit from it. It is targeted at all learners around the globe. However, learners from areas with limited resources have a special place in mind.

The book *"iEmergency Medicine for Medical Students and Interns"* is a part of the project content and service. It was created by 133 international contributors. At the publication time, the book content provided in the [iem-student.org](http://iem-student.org) has been visited by more than **20,000 visitors from 150 countries**.

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Arif Alper Cevik, MD, FEMAT, FIFEM

*Founder and Director, iEM Education Project*





# Preface



*“if you want to go fast go alone,  
if you want to go far go together”*

*African Proverb*

Undergraduate Emergency Medicine Education (UEME) is an undervalued area in the development of Emergency Medicine around the globe. If you read the articles regarding Emergency Medicine clerkships or if you travel to different countries and discuss their undergraduate education with local leaders, you can easily recognize the gaps between countries.

Today, there are few countries in the world that have appropriately designed UEME programs in their medical schools. The majority of the countries (even some developed ones) have no guidelines, curricula, or enough educational resources. In addition, there are limited resources (textbooks, websites) for medical students/interns which covers their educational needs based on current UEME recommendations.

This book is a product of an international collaboration of emergency physicians and Emergency Medicine enthusiasts. It intends to show that we can produce a free book and resource if we work collaboratively. It is a product of endless hours of hard work of all Editors, authors, and contributors. We thank all of them for trusting us in this journey.

This is just a start to build up better Emergency Medicine resources for medical students and interns, especially for developing countries. It is a continuous process, and there are a lot of areas that we need to improve in this book. Therefore, we are looking forward to your feedback and collaboration.

We also believe that international UEME will reach the minimum required standards in all countries based on the endless collaboration of emergency medicine professionals.

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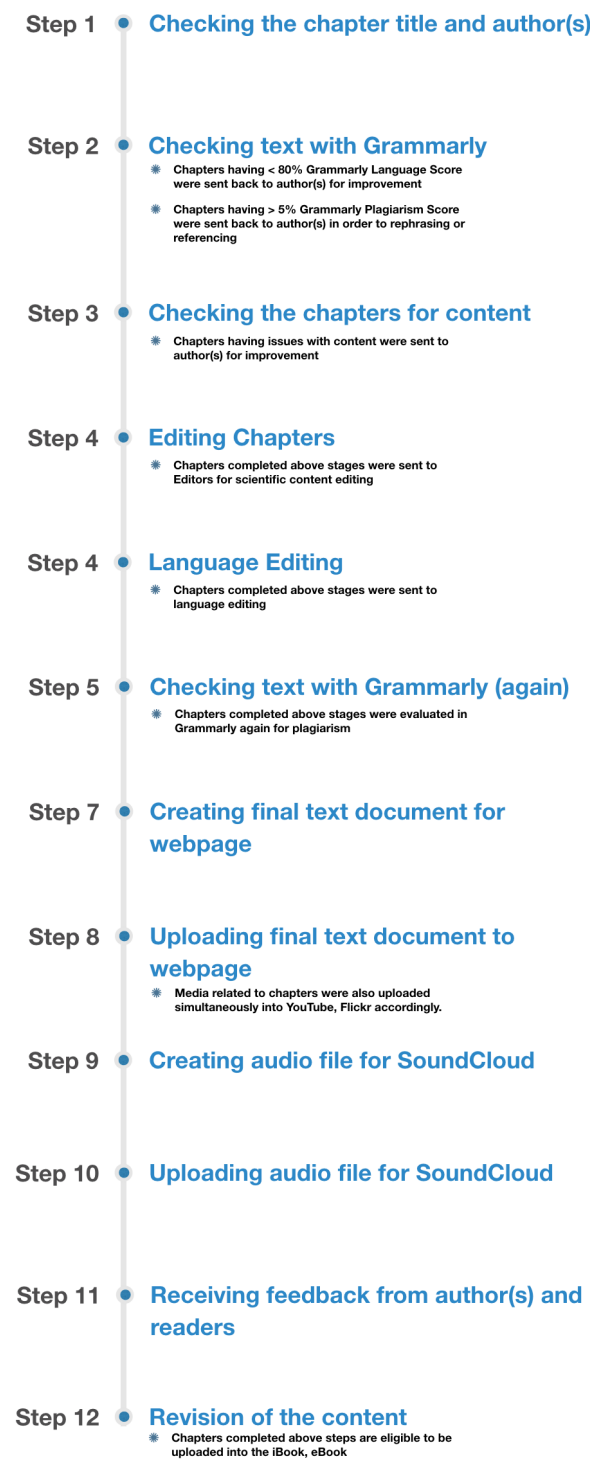
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# Publishing Process



There is continuous work for the iEM book process. We applied multiple editing and reviewing steps. We continue this process for many chapters with the feedback from our readers and contributors.

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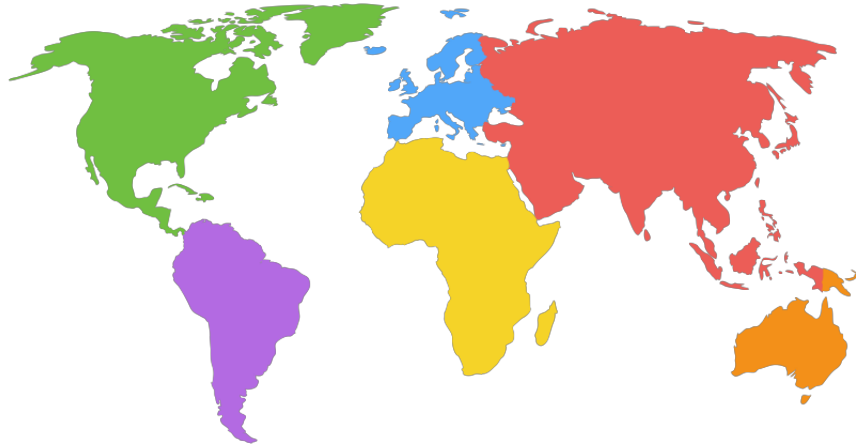
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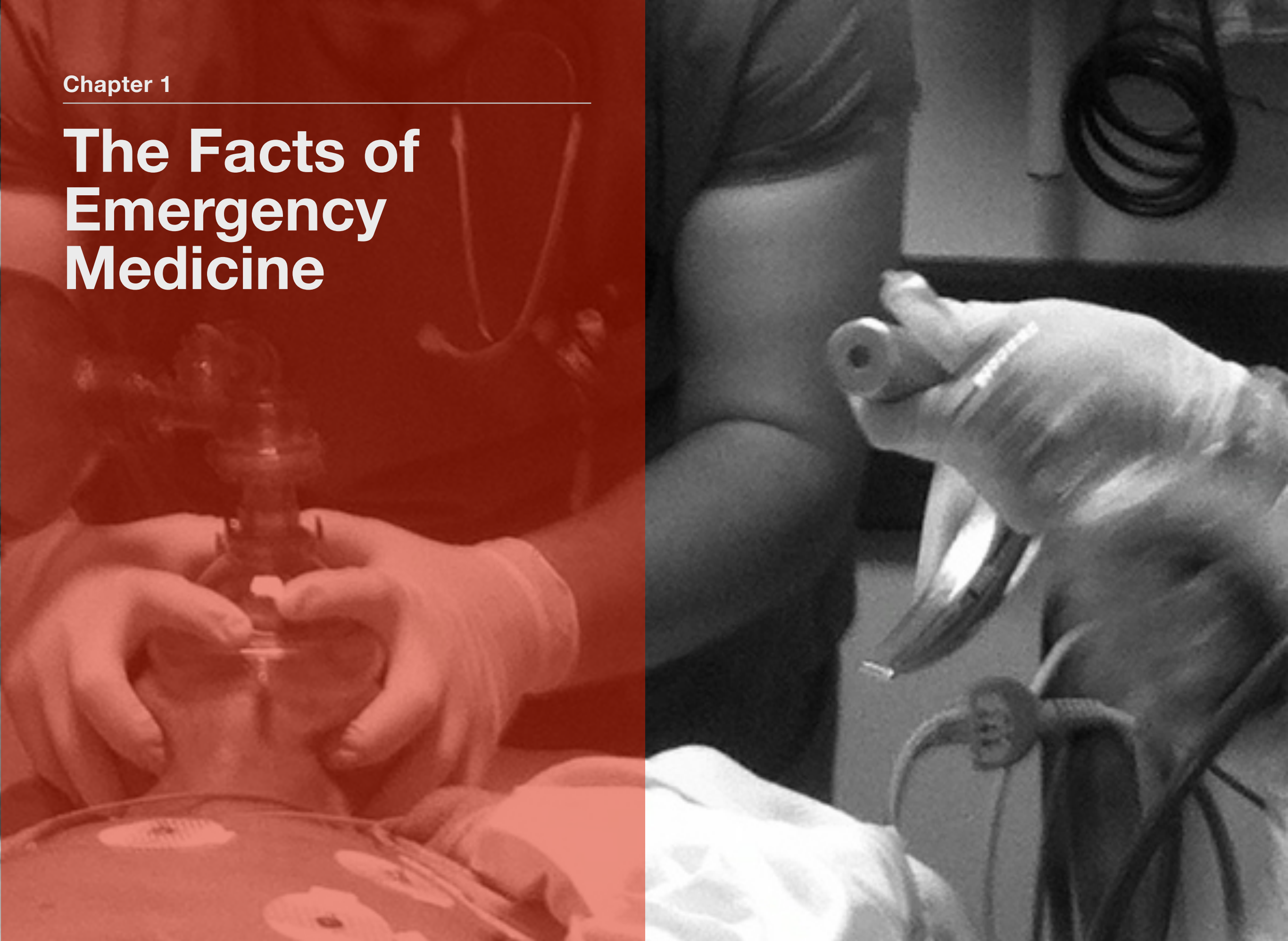
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*Sarah Attwa, Marwan Galal*

## Chapter 1

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# The Facts of Emergency Medicine



# Emergency Medicine: A unique specialty

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by Will Sanderson, Danny Cuevas, Rob Rogers

Imagine walking into the hospital to start your day – ambulances are blaring, the waiting room is clamoring, babies are crying. You stroll through this sea of humanity and eventually arrive at your workstation. After setting your bag down, you prepare the basic tools of your trade: a stethoscope, a fresh cup of coffee, and a sharp mind. Taking a deep breath, you prepare for the routine of yet another shift. But there is no “routine.” There is only the excitement and variety of what is about to come through those sliding double doors. That flimsy piece of metal and glass is the only barrier that separates you from the thousands of people with a multitude of medical ailments, any one of which could bring them to your doorstep. With a low hum and an almost silent whoosh, these doors part to reveal your next patient. To them, it is probably the worst day of their life. For you, it’s another Tuesday.

Who will be your next patient? Is it the 4-year-old boy with the asthma attack gasping for that next breath? Will it be the 78-year-old widower who fell at home while fixing himself a sandwich? Maybe it’s the 31-year-old female who just rear-ended another vehicle at highway speed; oh, did they also mention she’s 28 weeks pregnant? You look over and see new patients filling the critical examination rooms and the trauma bay. No matter what walks through that door, you’ll be ready. You sit down. You grab a chart. It’s time to get to work. Today is going to be another routinely awesome day.



Audio is available [here](#)



Why choose a career in emergency medicine? Before discussing where the field is going, it's important to know a little background on where it has been. And if you're reading this and considering a career in EM, do yourself a favor – take the time to watch [this documentary](#) from the Emergency Medicine Residents' Association ([EMRA](#)). As you'll see, the specialty of emergency medicine has evolved drastically over the last several decades and continues to be an increasingly popular choice among graduating medical students. Only a few decades ago, emergency departments around the country were staffed by physicians with a variety of training backgrounds. The vast majority of these physicians had little to no emergency medicine training at all. General surgeons, family physicians, neurologists, and even psychiatrists were among those that staffed emergency departments around the country and throughout the world. But since the establishment of the first emergency medicine residency programs in the 1970's and the subsequent establishment of the American Board of Emergency Medicine in 1979, the specialty has continued its rapid development in defining its place in the house of medicine. Walk into anything other than the smallest of EDs these days and you're likely to encounter an emergency medicine residency trained physician. A study published in 2008 demonstrated that in its relatively short history as a recognized medical specialty, the number of physicians staffing departments across the country who had received emergency medicine training soared from 0% to 70%. Why the dramatic shift? To understand the answer to this question, you need to take a

deeper look into the practice and lifestyle of the modern emergency medicine physician.

## Why EM?

Emergency medicine is a fast paced, team oriented, dynamic specialty that focuses on the rapid evaluation and treatment of a diversified patient population consisting of both pediatric and adult patients. As the initial provider for many of their patients, the emergency medicine physician is charged with the rapid assessment and data gathering needed to launch the initial workup and management of a wide variety of complaints that bring patients to the ED. Their work has an incredible influence in the patients' care as it generates the driving force for further medical evaluation; whether the patient is admitted to the hospital or discharged home, the emergency physician plays a huge role in directing both short and long term care well after their stay in the emergency department. Here's a look inside the lives of several emergency physicians from **Rob Orman** of [ERcast](#). Variety is the spice of the EM life. There is no set routine or expected patient list for the day. In the short span of a shift, you may diagnose strep pharyngitis, intubate an unresponsive patient who overdosed on heroin, reveal a cancer diagnosis to a young patient with flu-like symptoms, reduce a dislocated hip, place a chest tube in a patient with a hemothorax, and resuscitate a patient undergoing a cardiac arrest. Your next patient could be a six-year-old or a 75-year-old, both with abdominal pain. In a setting where some may see chaos, EM physicians find order. It's

exciting. It's energizing. This diversity is a uniquely challenging aspect of the medicine practiced in the emergency department.

EM physicians pull from a knowledge base that spans all medical specialties including pulmonology, cardiology, gastroenterology, trauma surgery, nephrology, ophthalmology, psychiatry, and neurology. Jack of all trades? Sure. Master of none? Not even close. The gap between the medical and surgical specialties is bridged within the practice of emergency medicine. The combination of a broad knowledge base with the need to develop a focused procedural skill set makes the EM physician a veritable Swiss Army Knife within the house of medicine. From endotracheal intubation, cricothyroidotomy, fracture reduction, and central line placement to pericardiocentesis, thoracotomy, chest tube placement, and lateral canthotomy, even the most enthusiastic proceduralist will find his hands full working in the ED.

Variety is a word that not only defines the practice of emergency medicine but also the lifestyle it affords. Are you a morning person who is up at the break of dawn and thinks best with a fresh mind after breakfast? Or are you a night owl who gets a burst of energy in the wee hours of the night when most others are sound asleep? Are you a weekend warrior who prefers to keep your schedule open on those days? Or would you rather work during the day to finish in time to pick up your children after they finish their day at school? Irrespective of your preference,

the shift work in the emergency department affords a level of flexibility not seen in other medical specialties. Emergency physicians manage the hustle and bustle of their department for a set number of hours, after which a fresh physician team arrives to take over. After his shift, the previous doctor hands over the care of his patients to the oncoming team to continue with the diagnostic and therapeutic management of the patient. In this regard, one can wrap up, sign out, and head home without bringing any of his work with him. The nature of shift work also allows for trading of shifts amongst the physicians staffing the department. Want a week off in April to spend some time at the beach? As long as you plan in advance, you shouldn't have any trouble getting there. With enough planning, it's quite possible to be at nearly every important life/family event you choose.

Within the field of emergency medicine, physicians are employed in several settings. These settings range from hospital-based and freestanding emergency departments, urgent care facilities, observation medicine units, emergency medical response services, and even telemedicine locations. Patient volumes, even at facilities in close proximity to one another, can vary greatly. Some facilities are designated trauma centers while others are not. There are facilities teamed up with a strong academic center to provide numerous subspecialty support and others are resource-limited community hospitals. No matter what your preference, there are a variety of settings to fit your needs. But let's get to the real question at hand: are emergency medicine



physicians satisfied with their career? This is really the crux of any discussion regarding career choice. How devastating would it be to realize after spending over a decade in college, medical school, and residency that working in the emergency department isn't for you? Well, in 2015, emergency medicine physicians came in 4th in overall career satisfaction compared to other medical specialties. 60% of all emergency physicians surveyed were satisfied with their income. Emergency physicians typically work more intensely for fewer total hours compared with other physicians and enjoy above-average compensation per hour. Below, Dr. Kevin King of the University of Texas Health Science Center San Antonio discusses the Pros and Cons of a Career in Emergency Medicine: [Pros and Cons of a Career in Emergency Medicine](#). As you can see, the life of an emergency medicine physician is not a perfect fit for everyone. EM physicians suffer from relatively high rates of burnout. However, as the field evolves and physician wellness becomes a priority for all physicians within medicine, this will surely improve. If the characteristics outlined above are consistent with the qualities you are looking for in a specialty, emergency medicine may well be the perfect fit for you.

**References and Further Reading**, click [here](#).

# Choosing the Emergency Medicine As A Career

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by C. James Holliman

The specialty of **Emergency Medicine** (EM) is a great career choice for medical students and interns. In August 2013, I celebrated my 30th year in full-time EM clinical practice, and I remain very happy and satisfied with my career choice. I have served as a career advisor to medical students and interns for over 30 years now and am very interested in encouraging people to undertake EM as a career.

Why is EM a great career? The main summary reason is that it is challenging and very personally rewarding. You can directly and quickly see the benefits and positive results of your diagnosis and treatment of patients who have emergent medical conditions. You have the satisfaction of knowing you have made a big positive difference in patients' lives and well-being. EM encompasses a very wide variety of patients and medical and surgical problems. EM deals with patients of both genders and all ages. The variety of cases seen by EM is probably greater than that of any other specialty, and this aspect is part of what makes EM so interesting and stimulating. The practice of EM encompasses a nice mix of diagnostic medicine and of performing diagnostic and therapeutic procedures. The EM practitioner sees patients with undifferentiated symptoms and so must make the initial diagnosis of many conditions. EM interacts with all the other medical specialties, and at most hospitals accounts for the majority of hospital admissions.

 A video is available [here](#)

 Audio is available [here](#)

Unique subjects routinely taught in EM include: cost-effective ancillary test ordering, efficiency in patient flow, managing multiple simultaneous patients, coordinating Prehospital and Emergency Department (ED) care, focusing the approach to medical problems, speed and efficiency of patient evaluations, efficient use of ancillary personnel, efficient recording and transmittal of clinical data, and injury and violence prevention. EM is also a young, vibrant specialty with a lot of enthusiastic practitioners, most of whom have extended interests and talents outside of medical practice, and who serve as role models and mentors.

EM also encompasses the supervision of and interactions with prehospital care. EM receives patients brought to the hospital ED by ambulance. EM is responsible for training the prehospital personnel, and in some countries, EM practitioners may find themselves directly staff ambulances, both ground and aeromedical. EM also is the main specialty involved in the planning for, and management of disasters and mass casualty situations, both of which also require close interaction with prehospital care.

EM has well-developed residency training programs for medical school graduates in many countries. The length of these training programs varies from country to country but generally is three to five years. The EM residencies each have some clinical rotations on other services or specialties (such as anesthesia, surgery,

intensive care, pediatrics, obstetrics, internal medicine, cardiology, trauma, etc.), and this direct exposure to other multiple specialties makes EM residencies more interesting. Of course, the majority of time in most EM residencies is spent in the hospital ED. Most EM residencies also offer opportunities to participate in prehospital care and EM research. One validation of the strength of EM as a career is that in the U.S. each year it is the first or second most popular choice for residency by medical students, and the overall residency program “fill” rate in the National Residency Matching Program is over 99%. For more information on EM residencies from the perspective of EM residents, check the website [www.emra.org](http://www.emra.org).

EM has also developed a number of sub-specialties which enhance the career options in EM. Each sub-specialty offers post-residency fellowship training programs of one to three years duration. In the U.S.A., the following EM subspecialties are officially recognized and have their own sub-specialty exam certification: Pediatric EM, Toxicology, Critical Care, Sports Medicine, Hyperbaric Medicine, Emergency Medical Services, and Palliative Care. Additional EM sub-specialty fellowship programs include International EM (or Global Health), Ultrasound, Research, Education, Simulation Training, Aeromedical, Disaster, Trauma, Administration, and Information Technology. Check the website [www.saem.org](http://www.saem.org) for the most up to date listing of EM fellowship programs.

One of the greatest assets of EM as a career is the wide variety of post-residency career choices or options. These include practicing in a variety of hospital types: university, teaching, community, government, military, etc. EM physicians can also practice in “freestanding” ED’s (not directly connected to a hospital) or in urgent care centers. If a person does not want to practice at just one hospital, there is the option to undertake “locum tenens” practice in which the person works clinical shifts at multiple different facilities. For those in the military or interested in a military career, EM has been shown to be one of the most needed specialties in the military. EM physicians can undertake leadership positions in hospital administration, prehospital care, and in the government developing and directing health policy.

Unique advantages of EM as a career include the almost unlimited opportunities in international EM development, control over and predictability of one’s work schedule, usually not having to be “on-call” when not directly on duty, and having “geographic flexibility” in the variety of places to practice. There are also a relatively small number of EM physicians in academic practice, so it is often easy for physicians interested in an academic career to rapidly advance up the “academic ladder”.

Another positive aspect for EM is that in most countries it is projected to be an undersupplied specialty for many years, and so there will continue to be many open job opportunities in EM. EM has also been shown to be a critical component of any

national healthcare system, and there is extensive medical literature support for the value and efficacy of EM.

Another nice EM career aspect is the opportunity after residency or fellowship to participate in one or more of the EM specialty state, national, regional, or international organizations. Most countries have a national EM organization which carries out some activities including annual educational conferences. The International Federation for EM (IFEM) has a large number of committees, task forces, and special interest groups which are carrying out a wide variety of projects which need more individuals to participate and contribute (check [www.ifem.cc](http://www.ifem.cc) for more information on IFEM). Becoming involved with one or more of the EM specialty organizations can provide one with career satisfaction in helping improve and develop the specialty as well as obtaining leadership training and experience.

So in summary, EM is a great career choice with a very wide range of post-residency work options, a very safe job market for the future, and the personal satisfaction of knowing one’s work directly and quickly helps patients, and that one’s work is a critical component of the national healthcare system.

**References and Further Reading**, click [here](#).

# Thinking Like an Emergency Physician

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by Joe Lex

*“Emergency Medicine is the most interesting 15 minutes of every other specialty.”*

– Dan Sandberg, BEEM Conference, 2014

Why are we different? How do we differentiate ourselves from other specialties of medicine? We work in a different environment in different hours and with different patients more than any other specialty. Our motto is “Anyone, anything, anytime.”

While other doctors dwell on the question, “What does this patient have? (i.e., “What’s the diagnosis?”), emergency physicians are constantly thinking “What does this patient need? Now? In 5 minutes? In two hours?” Does this involve a different way of thinking?

The concept of seeing undifferentiated patients with symptoms, not diagnoses, is alien to many of our medical colleagues. Yet we do it on a daily basis, many times during a shift. Every time I introduce myself to a patient, I never know which direction things are going to head. But I feel like I should give the following disclaimer. Hello stranger, I am Doctor Joe Lex. I will spend as much time as it takes to determine whether you are trying to die on me and whether I should admit you to the hospital so you can try to die on one of my colleagues. You and I have never met before today. You must trust me with your life and secrets, and I must trust that the answers you give me are honest. After today, we will probably never see one another again. This may turn out to be one of the worst days of your life; for me, it is another workday. I may forget you minutes after you leave the department, but you will probably remember me for many months or years, possibly even for the rest of your life. I will ask you many, many questions. I will do



Audio is available [here](#)



the best I can to ask the right questions in the right order so that I come to a correct decision. I want you to tell me the story, and for me to understand that story, I may have to interrupt you to clarify your answers. Each question I ask you is a conscious decision on my part, but in an average 8 hour shift I will make somewhere near 10,000 conscious and subconscious decisions – who to see next, what question to ask next, how much physical examination should I perform, is that really a murmur that I am hearing, what lab study should I order, what imaging study should I look at now, which consultant will give me the least pushback about caring for you, is your nurse one to whom I can trust the mission of getting your pain under control, and will I remember to give you that work note when it is time for you to go home? So even if I screw up just 0.1% of these decisions, I will make about ten mistakes today. I hope for both of our sakes you have a plain, obvious emergency with a high signal-to-noise ratio: gonorrhea, a dislocated knee cap, chest pain with an obvious STEMI pattern on EKG. I can recognize and treat those things without even thinking. If, on the other hand, your problem has a lot of background noise, I am more likely to be led down the wrong path and come to the wrong conclusion. I am glad to report that the human body is very resilient. We as humans have evolved over millennia to survive, so even if I screw up, the odds are very, very good that you will be fine.

Voltaire told us back in the 18th century that “The art of medicine consists of amusing the patient while nature cures the disease.”

For the most part, this has not changed. And Lewis Thomas wrote: “The great secret of doctors, learned by internists and learned early in marriage by internists’ wives, but still hidden from the public, is that most things get better by themselves. Most things, in fact, are better by morning.” Remember, you don’t come to me with a diagnosis: you come to me with symptoms.

You may have any one of more than 10,000 diseases or conditions, and – truth be told – the odds of me getting the absolute correct diagnosis are not good. You may have an uncommon presentation of a common disease or a common presentation of an uncommon problem. If you are early in your disease process, I may miss such life-threatening conditions as heart attack or sepsis. If you neglect to truthfully tell me your sexual history or use of drugs and alcohol, I may not follow through with appropriate questions and come to a totally incorrect conclusion about what you need or what you have.

The path to dying, on the other hand, is rather direct – failure of respirations, failure of the heart, failure of the brain, or failure of metabolism.

You may be disappointed that you are not being seen by a “specialist.” Many people feel that when they have their heart attack, they should be cared for by a cardiologist. So they think that the symptom of “chest pain” is their ticket to the heart specialist. But what if their heart attack is not chest pain, but nausea and breathlessness; and what if their chest pain is aortic

dissection? So you are being treated by a specialist – one who can discern the life-threatening from the banal, and the cardiac from the surgical. We are the specialty trained to think like this.

If you insist on asking “What do I have, Doctor Lex?” you may be disappointed when I tell you “I don’t know, but it’s safe for you to go home” without giving you a diagnosis – or without doing a single test. I do know that if I give you a made-up diagnosis like “gastritis” or “walking pneumonia,” you will think the problem is solved, and other doctors will anchor on that diagnosis, and you may never get the right answers.

Here’s some good news: we are probably both thinking of the worst case scenario. You get a headache and wonder “Do I have a brain tumor?” You get some stomach pain and worry “Is this cancer?” The good news is that I am thinking exactly the same thing. And if you do not hear me say the word “stroke” or “cancer,” then you will think I am an idiot for not reading your mind to determine that is what you are worried about. I understand that, no matter how trivial your complaint, you have a fear that something bad is happening.

While we are talking, I may be interrupted once or twice. See, I get interrupted several times every hour – answering calls from consultants, responding to the prehospital personnel, trying to clarify an obscure order for a nurse, or I may get called away to care for someone far sicker than you. I will try very hard to not let

these interruptions derail me from doing what is best for you today.

I will use my knowledge and experience to come to the right decisions for you. But I am biased, and knowledge of bias is not enough to change my bias. For instance, I know the pathophysiology of pulmonary embolism in excruciating detail, but the literature suggests I may still miss this diagnosis at least half the time it occurs.

And here’s the interesting thing: I will probably make these errors whether I just quickly determine what I think you have by recognition or use analytical reason. Emergency physicians are notorious for thinking quickly and making early decisions based on minimal information (Type 1 thinking). Cognitive psychologists tell us that we can cut down on errors by using analytical reasoning (Type 2 thinking). It turns out that both produce about the same amount of error, and the key is probably to learn both types of reasoning simultaneously.

After I see you, I will go to a computer and probably spend as much time generating your chart as I did while seeing you. This is essential for me to do so the hospital and I can get paid. The more carefully I document what you say and what I did, then the more money I can collect from your insurance carrier. The final chart may be useless in helping other health care providers understand what happened today unless I deviate from the clicks and actually write what we talked about and explained my

thought process. In my eight hour shift today I will click about 4000 times.

What's that? You say you don't have insurance? Well, that's okay too. The US government and many other governments in the world have mandated that I have to see you anyway without asking you how you will pay. No, they haven't guaranteed me any money for doing this – in fact, I can be fined a hefty amount if I don't. A 2003 article estimated I give away more than \$138,000 per year worth of free care related to this law.

But you have come to the right place. If you need a life-saving procedure such as endotracheal intubation or decompression needle thoracotomy, I'll do it. If you need emergency delivery of your baby or rapid control of your hemorrhage, I can do that too. I can do your spinal tap, I can sew your laceration, I can reduce your shoulder dislocation, and I can insert your Foley catheter. I can float your temporary pacemaker, I can get that pesky foreign body out of your eye or ear or rectum, I can stop your seizure, and I can talk you through your bad trip.

Emergency medicine really annoys a lot of the other specialists. We are there 24 hours a day, 7 days a week. And we really expect our consultants to be there when we need them. Yes, we are fully prepared to annoy a consultant if that is what you need.

I have seen thousands of patients, each unique, in my near-50 years of experience. But every time I think about writing a book

telling of my wondrous career, I quickly stop short and tell myself “You will just be adding more blather to what is already out there – what you have learned cannot easily be taught and will not be easily learned by others. What you construe as wisdom, others will see as platitudes.”

As an author, Norman Douglas once wrote: “What is all wisdom save a collection of platitudes. Take fifty of our current proverbial sayings– they are so trite, so threadbare. None the less they embody the concentrated experience of the race, and the man who orders his life according to their teachings cannot be far wrong. Has any man ever attained to inner harmony by pondering the experience of others? Not since the world began! He must pass through fire.”

Have you ever heard of John Coltrane? He was an astonishing musician who became one of the premiere creators of the 20th century. He started as an imitator of older musicians but quickly changed into his own man. He listened to and borrowed from Miles Davis and Thelonious Monk, African music and Indian music, Christianity and Hinduism and Buddhism. And from these disparate parts he created something unique, unlike anything ever heard before. Coltrane not only changed music, but he altered people's expectations of what music could be. In the same way, emergency medicine has taken from surgery and pediatrics, critical care and obstetrics, endocrinology and psychiatry, and we

have created something unique. And in doing so, we altered the world's expectations of what medicine should be.

*Now, how can I help you today?*

**References and Further Reading**, click [here](#).



## Chapter 2

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# Emergency Medicine Clerkship: Things to Know





# The Importance of The Emergency Medicine Clerkship

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by Linda Katirji, Farhad Aziz, Rob Rogers

## Introduction

The Emergency Medicine (EM) clerkship typically takes place during the fourth year of medical school. However, some programs may have an optional elective during the third year. Whether or not you plan to specialize in Emergency Medicine, the rotation is an important aspect of your medical education. The emergency room is a unique learning environment which is different than any other setting in the hospital. It provides clinical opportunities that are largely unavailable in other clerkships and rotations. During residency, many specialties will also spend a significant amount of time in the Emergency Department (ED). This may be within a structured EM rotation, or while admitting or seeing patients for a certain medical or surgical service. Therefore, it is important to gain an understanding of the flow of the ED as well as the particular thought process that must be employed with emergency department patients. This chapter will discuss some of the unique aspects of the emergency department, some of the skills to acquire during the EM clerkship, as well as how to best be successful and take the most away from your rotation.

## Unique Aspects of The Emergency Department Environment

The high volume and acuity of patients in the ED create a time pressure and forces physicians to employ a different style of practice than in other settings. A steady



Audio is available [here](#)

stream of patients, some of whom may require immediate life-saving measures, means that many times there is little to no time to review history or any medical records prior to seeing the patient. A majority of the time you will need to assess a patient without knowing anything about their background. Therefore, it is important to gain an understanding of what the most important pieces of information to gather are for each patient. This can be difficult since most patients will arrive with completely undifferentiated complaints. Some common examples of these undifferentiated complaints are “chest pain” and “abdominal pain”, where the etiology can range from completely benign to immediately life-threatening, or “weakness”, where the differential diagnosis includes essentially the entire spectrum of medical pathology.

This undifferentiated patient is the standard in the ED. However, they can present in any medical setting. It is important to learn the thought process and develop a strategy for thinking through these types of patients whether or not you plan on a career in EM. Emergency Physicians (EPs) must employ and master a completely different style of practice than most physicians. EPs must always think worst case scenarios for each chief complaint and must be knowledgeable and comfortable with associated the workup and treatment. A good example of this is chest pain. Even though many times the complaint of “chest pain” is found to be caused by a non-acutely life threatening etiology, EPs must immediately think of six fatal causes of chest pain: acute coronary

syndromes, aortic dissection, pulmonary embolism, pericardial tamponade, pneumothorax, esophageal rupture. Additionally, ED doctors have to use a different thought process in determining the disposition, or outcome, of the patient. The ED doctor essentially wants to avoid sending a patient home that should not go home, where as a consultant, or admitting service, does not want to admit a patient who shouldn't be admitted. This may seem trivial however this difference in thought process can occasionally create tension between the ED and admitting services.

Teaching in the ED is different than most other settings in the hospital as well. There is usually no time set aside for formal rounds, so most teaching is done at the bedside or at the time the student or resident presents the patient. Many times, attending physicians will pick out “teaching points” for each patient. Each physician will have a different teaching style, and your learning will, in general, be more active than passive.

Lastly, the ED is a great place for medical students and first-year residents to learn to take responsibility for their patients. Students often time have an increased level of autonomy compared to other rotations. Many times, the student will be the first person to assess the patient, which is a very important role. It is important to learn to distinguish whether a patient is “sick” or “not sick”, and whether or not at first glance you think this patient could go

home or needs to be admitted not matter what the diagnosis may be.

## Unique Skills To Take Away From EM Clerkships

Emergency medicine is a wonderful rotation that exposes you to different patient populations but also a variety of pathology. This diverse collection of patients and pathology lends emergency medicine residents and students a unique opportunity to gain a mastery of different skills. These skills range from a knowledge of how to approach critically ill patients, gaining procedural skills, reading radiographs and CT scans and performing ultrasounds and much more.

Often you may be busy doing different tasks when you have to drop everything and manage a critically ill patient. This is one of the exciting aspects of emergency medicine. These patients offer students a great opportunity to learn the principles of resuscitation, such as managing airways and circulatory collapse, identifying causes for the patient's decompensation, and instituting the appropriate treatment. Whether you pursue a career in emergency medicine or choose to pursue a different specialty, critically ill patients will always be a part of your patient population. Understanding how to approach and stabilize these patients is an important part of being a physician.

Though learning the art of resuscitation is a vital part of the EM rotation, this is also an opportunity to gain competence in a

variety of procedures. Whether you intend to pursue a career in pediatrics, internal medicine, orthopedics, general surgery or any other specialty, your rotation through the ED will expose you to a wide array of procedural skills ranging from intubating and placing central lines and arterial lines in the critically ill to performing lumbar punctures and fracture reductions in children. Autonomy is encouraged with procedures, and you will have the opportunity to improve your skills and techniques under the guidance of residents and attendings. EM is a very hands on specialty. You should take advantage of medical student and resident didactics as a which may include procedure labs on mannikins or cadavers and simulation. This will give you an opportunity to practice and provide better patient care during your rotation.

In addition to becoming familiar with a wide array of procedures, your EM clerkship will also allow you to familiarize yourself with a variety of imaging modalities ordered in the ED. There is a tremendous amount of pathology found in the ED which lends itself to a range of imaging. Whether it be learning to perform bedside ultrasonography on a crashing patient or simply learning how to approach a chest x-ray or a CT scan of the abdomen, your EM rotation will give you plenty of opportunities to become proficient in a skill you will need later in your career.

Though your EM clerkship gives you exposure to a tremendous amount of skills which will help you become a savvy doctor, no

skill is more important than compassion and humility. Everyday you will meet patients on the worst day of their life. Realizing this and comforting them and their families is paramount to your success as a physician. You will also encounter a variety of consulting physicians. Some are nice and professional while others are not. Having a general understanding that they all have knowledge that you can learn from will set you up for a successful career in medicine.

## How To Be Successful on Your EM Clerkship

Many of the of the same qualities that allow you to be successful in other rotations will help you to be successful in the ED. It is important to be hardworking, proactive, and knowledgeable. Keep a close eye on your patients, re-evaluate them frequently, and make sure to follow up on any results, including labs, imaging, and any recommendations by consultants. The unique aspects of the ED and EM clerkship discussed previously mean the first few shifts may be stressful and seem hectic. For every student and resident that rotates through the ED there is a significant learning curve – with each shift you spend in the ED, things will feel less and less daunting. It's important during this time that you know your limitations and what you are comfortable and not comfortable with. Many times you will be the first person to assess the patient. You should have a low threshold for alerting an upper-level resident or attending if the patient appears to be sick, or if they present with a complaint you are uncomfortable with. At the same time, you should be confident in what you do

know and take the opportunity to learn how to diagnose, treat, and manage your patient.

The best way to build confidence during your EM rotation is to gain experience and knowledge. Try to be proactive in learning new procedures or treatments with attending or resident assistance. Additionally, it's very important to keep up with reading and studying. In the Emergency Room, you may see medical pathology you've only read about and will be expected to know how to diagnose and treat these diseases properly.

Good communication is essential for a physician in any specialty, and in the ED, it is an imperative skill to have. You will be working with a large team of nurses, technicians, consulting doctors, social workers, and paramedics, just to name a few. When you see a patient, it is a good idea to speak with the nurse before you enter the room to gain a better understanding of the patient's complaint, as well as gather any information that was relayed by EMS. By communicating the plan of care to the nurse and supporting staff, you will not only improve patient care and reduce mistakes but also forge relationships that will enrich your experience in the ED. In acute settings such as a patient code or rapidly decompensating patient, good communication with the entire team is critical. As a medical student or rotating resident, this is a great time to practice and improve your communication skills in these acute settings under the direction of residents and attendings.

Your EM rotation will be an exciting, unique experience during medical school and residency. Whether you plan to specialize in EM or not, you will learn many procedural skills, improve your own method of diagnosing and treating patients and be able to practice a different method of medical decision making.

**References and Further Reading**, click [here](#).



# Medical Professionalism

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by Amila Punyadasa

*“The Dimensions That All Medical Students Should Know About”*

## Introduction

It is prudent to commence this chapter with some relevant definitions. A profession is a specific type of occupation, one that performs work with special characteristics while competing for economic, social, and political rewards. A professional, it follows, is a person who belongs to a group (profession) which possesses specialized characteristics (specifically, knowledge, skills, and attitudes) that have been obtained after a long period of study and are used to benefit other members of society. Thus, professionalism is used to describe those skills, attitudes and behaviors. We expect from individuals during the practice of their profession and includes such concepts as maintenance of competence, ethical behavior, integrity, honesty, altruism, service to others, adherence to professional codes, justice, respect for others and self-regulation. In fact, it has been said that professionalism serves as the basis of the medical professions' relationship to society and that this relationship is a social contract, underpinned by professionalism. Medical professionalism thus comprises physicians' behaviors that demonstrate they are worthy of the trust the public and patients place in them.

In essence, our profession involves healing. Kirk (2007) eloquently stated that in any patient encounter, we consider both a right and good healing strategy for our patients. The right action is informed by evidence-based medicine, while the good

action incorporates the patient's values and preferences aligned with the physician's judgment (or, in other words, requires the physician to exercise various dimensions of professionalism).

Interestingly, this 'judgment' itself has three concrete steps:

1. The diagnostic question – What is wrong with the patient? – Incorporates both the clinical assessment and investigations the patient was subjected to.
2. The therapeutic question – What can be done for the patient? – Informed by evidence and may involve a plethora of treatments and interventions.
3. The prudential question – What should be done for the patient? – This involves the patient in the final decision-making process, preserves patient autonomy, and ensures a patient-centric approach to healthcare provision.

The advantages of teaching students to practice professionalism include imparting a greater sense of purpose, building a framework for harmonious and efficacious healthcare provision, as well as building trust and mutual respect. Additional benefits include improving patient satisfaction, reducing complaints and litigation, improving treatment compliance, and improving clinical outcomes.

Although there are many definitions of professionalism, most contain a list of responsibilities that the physician should exercise; they are based on three fundamental principles:

1. The principle of primacy of patient welfare – This is based on a dedication to serving the interest of the patient with altruism; in turn, contributing to trust, the core of the doctor-patient relationship. This principle should be unsullied by economic, administrative or sociocultural exigencies.
2. The principle of patient autonomy – The patient's autonomy must be respected, and doctors should not only be honest with their patients but also empower them to make informed decisions about their healthcare, as long as these are within the ethical practice and do not lead to demands for inappropriate care.
3. The principle of social justice – This includes the fair distribution and access to health care resources and the elimination of discrimination, whether that be racial, gender-based, religious, socio-economic or any other social category.

The definition proposed by Wilkinson et al. (2009) is both thorough and explicit. His list of specific behaviors necessary for medical professionalism is enumerated. Also incorporated in the appendix is the Emergency Medicine Clerkship's core curriculum learning objectives that pertain to professionalism.

- Honesty/Integrity

- Confidentiality
- Moral reasoning and ethical decision making
- Respecting privileges and codes of conduct
- Effective Interactions with Patients and Their Relatives
  - Respect for diversity
  - Politeness/Courtesy/Patience
  - Manners/ Demeanor
  - Patient-centered and involved decision-making process
  - Maintenance of professional boundaries
  - Balancing availability to others with care of ones-self
- Effective Interactions with Other Health Care Workers
  - Teamwork
  - Respect for diversity
  - Politeness/Courtesy/Patience
  - Maintenance of professional boundaries
  - Manner/Demeanor – This includes maintaining a professional appearance.

- Balancing availability to others with care of ones-self
- Reliability
- Accountability/task completion
- Punctuality
- Assumes responsibility and is conscientious
- Self-Reflectiveness
- Recognizing limits
- Life-long learning
- Dealing with uncertainty
- Teaching and debriefing
- People management
- Leadership
- Using appropriate strategies to improve processes
- Advancing knowledge and one's field (e.g. via Research)

Seven deadly sins of professionalism or professional non-virtues.  
They are the following:

1. Greed – with respect to money, power, and fame.

2. Abuse of power – with respect to colleagues, patients, and position in the hierarchy.
3. Arrogance – towards patients or colleagues
4. Conflict of Interest
5. Misrepresentation – for example, lying or being fraudulent.
6. Apathy – pertaining to lack of commitment, irresponsibility or doing the bare minimum for patients.
7. Impairment – secondary to illness, alcohol or drugs.

Many of the core criteria of professionalism are related to the assimilation of good old-fashioned virtues and the development of soft skills that must not only be taught but also reinforced through modeling and active practice. When devising any professional curriculum, take careful consideration of common themes that positively impact necessary behavioral changes. Some of those themes include the following:

1. Motivation (or Getting “Buy-In”)
  - a. Intrinsic – Medical students must be convinced of the importance of the desired change in behaviors for it to be a driving force.
  - b. Extrinsic – The principal extrinsic motivator for medical students is the knowledge that professionalism will not only be

explicitly taught but also explicitly tested (or assessment driven learning and practice). Other motivators include bestowing rewards and recognition for demonstrating positive behaviors, a form of positive reinforcement.

2. Observing role models are of the utmost importance. This entails not only the incorporation of good behaviors observed but also the recognition of negative behaviors exhibited by poor role models and purposefully not engaging in such behaviors. I shall delve into this concept in more detail in part 4 below.
3. Continued exposure to aspects of professionalism is important to inculcate its tenets. Vertical integration into a spiral curriculum, in my opinion, is required to achieve this goal.
4. Reflection and feedback are keys. Self-reflection on aspects of professionalism, as well as timely and effective feedback of specific behaviors from peers and seniors, will help mold the medical student into doctors that demonstrate professional behaviors. Good feedback, based on observable behaviors, explains not only what should be done but also why it should be done, and both are essential for effective learning.

The importance of teaching medical professionalism to undergraduates is well documented in the literature and is integral to the medical profession. What is perhaps less clear is exactly how this teaching should be conducted. The solution lies in

understanding how to utilize all aspects of the curriculum including the formal, informal, and hidden curriculum.

## Strategies for Teaching the Formal Curriculum

The following involves a discussion of the teaching of the “formal curriculum,” which is defined as the stated, intended, and formally offered and endorsed curriculum.

Maudsley & Strivens (2004) have proposed that the ‘situated learning’ theory seems to describe the most effective model to imbibe the virtues of professionalism. It suggests that learning should be embedded in authentic activities which help to transform knowledge from the abstract and theoretical to the usable and useful. Brown et al. (1989) further noted that there should be a balance struck between the explicit teaching of a subject and activities where the implementation of such knowledge is utilized in an authentic context.

Furthermore, there have been two principle approaches described in the teaching of professionalism.

- Explicitly Teaching the Basics of Professionalism
- Experiential Learning

One should utilize both of these approaches. In fact, to paraphrase the situated learning theory, a balance must be struck

between knowledge-based teaching and in-situ experiential learning.

In my personal undergraduate and postgraduate training years, there has been a scarcity of teaching about professionalism, especially in the formal curriculum. Professionalism is not a gimmick but a set of rules and behaviors that each of us must adhere to and practice; it forms the very foundation of good clinical practice. Over the last 15 years, the undergraduate curriculum has increasingly incorporated medical professionalism, explicitly, as part of their formal curriculum; they do so with the application of both vertical and horizontal integration. This is the way forward.

The judicious instructor has a plethora of instructional modalities to choose from. The different teaching/learning strategies and tools that may be utilized to achieve competence across the many dimensions of medical professionalism successfully.

- Problem-based reflective practice
- Role-modeling
- Portfolio based training
- Clinical contacts with tutor debriefs
- Simulation-based training
- Didactics and tutorials



## The Informal and Hidden Curricula in Medical Professionalism

The Informal and hidden curricula are, as the terms themselves suggest, not only cloaked in mystery but are also crucial elements of medical professionalism. The informal curriculum is defined as an unscripted and ad hoc yet highly interpersonal form of teaching and learning that takes place between faculty members and students in non-classical teaching settings. Examples of these 'settings' could include tearooms during clinical breaks or even a certain coffee house. Over coffee, I recently taught my 4th-year elective student about medical professionalism themes, including respect for the patient and their autonomy, citing anecdotes from my experience.

The hidden curriculum, on the other hand, is a set of influences that function at the level of organizational structure and culture. These two components are interrelated; In fact, some authors refer to both the informal and hidden curricula as one entity. Hence, it is clear that in order to acquire the knowledge, skills, and attitudes of professionalism, students must use a wide range of learning strategies that extend far beyond the intended formal curriculum. These strategies include interactions with teachers, colleagues and various other people around them.

Research has shown a distressing downtrend in professionalism, which has been directly attributed to the influence of a hidden

curriculum. For example, empathy among medical students was seen to decrease as they progress through medical school.

Such surprising findings are noted despite increased emphasis being placed on the teaching of the formal curriculum. Thus, it becomes clear that these discrepancies may be attributed to the influence of the 'hidden' curriculum. Interestingly, some students believe that certain components of their learning could only be achieved through the informal and hidden curriculum and that the science of medicine is associated mainly with the formal curriculum while the art of medicine is associated mainly with the informal and hidden curricula.

It is well known that medical students acquire soft skills such as communication techniques and medical etiquette, both important facets of professionalism, from observing mentors, peers, and other healthcare workers.

Other modalities of learning via the informal and hidden curriculum are

- Rituals
- Infrastructure
- Chance Observations

It is quite clear to me that the formal, informal and hidden curricula are all complimentary. However, there is, unfortunately,

an ongoing conflict between the formal curriculum and the informal/hidden curriculum.

An obvious remedy is to engage the various stakeholders involved in training medical students in a constructive dialogue on how the hidden and informal curricula can be manipulated to influence student learning positively. This understanding will not only help avoid the visible conflict between formal curriculum and informal/hidden curricula but will also extract the advantages of the informal/hidden curriculum to produce better physicians.

It is undeniable that medical school faculty, both senior and junior doctors, and other healthcare workers are all role models who may influence medical students' learning. The professionalism demonstrated by all these people is of great importance not only for their patients but also for the next generation of doctors. Hence, we have to keep paramount in our minds that our practice and interpretation of professionalism, and all its dimensions, is keenly being observed by our students and that we have a huge role to play in the development and molding of their moral and professional wellbeing.

## **Role Modelling in Medical Professionalism**

This connects back to the most powerful tool to teach professionalism, role modeling. Role modeling involves a physician (or role model) who teaches a student by example; its importance is unquestionable and has been documented for many years.

Classically, a role model is someone who is admired for the way he acts and for his professionalism and whose behavior is considered as a standard of excellence to aspire to.

It is important to show students what right practice is, and that applies to both clinical and professional conduct. This is the essence of role modeling.

Paice et al. (2002) described the act of being a role model as serendipitous, a beneficial but chance outcome. I respectfully disagree. Senior tutors and physicians all act as role models and must be cognizant of everything we do in front of our students. Knowing that we will be observed and scrutinized should make us ultra self-conscious, and we should try hard to showcase and inculcate the virtues of sound clinical practice and professionalism at every opportunity.

The vast majority of the literature is in agreement that role modeling is not only important but also integral to medical education. Role models not only affect the attitudes, behaviors, and ethics of medical students but also imbibe professionalism in trainees. I am sure we can all recall a specific role-model that impressed upon us the virtues of professionalism while demonstrating punctuality, responsibility, honesty, ethical reasoning, accountability, collegiality and patient-centric management while embracing diversity with a sense of decorum. Such role models also influence career choices of students and function in the formal, informal and hidden curricula. However,

drawbacks have also been described. Sinclair (1997) wrote that he noted medical students being drawn to and indeed emulate senior doctors who held positions of responsibility and status. He further noted a warning of their professional ideals and behaviors as they evolved.

## Assessment Techniques in Medical Professionalism

Unfortunately, despite the unquestionable importance of professionalism to the everyday functioning of every medical doctor and student, my experiences (spanning two decades and three countries) with its assessment has been rather limited. In fact, during my postgraduate years of clinical practice, the assessment of professionalism has been rather rudimentary, with its evaluation often subordinate to the assessment of clinical competencies.

If we are to take the assessment of professionalism seriously, then we must improve our framework for assessment. Specifically, we need to implement a number of different methods to effectively measure all levels of Miller's pyramid, while also covering the multidimensional breadth of professionalism.

I shall now consider some assessment tools that will enable the ability to assess the multidimensionality of medical professionalism. These are;

- Assessment of an Observed clinical encounter

- Collated views of co-workers
- Simulation
- Paper tests
- Patient opinions
- Ratings by a Superior
- Self-assessment
- Critical incident report / Records of incidents of unprofessionalism

## Social Media and Professionalism

It seems like nearly everyone, certainly from the Generations Y and Z, is using Facebook or Twitter these days for one reason or another. Although not a fan myself, I do concede that when used with prudence, social media and the Internet is an invaluable resource for teaching and learning. It can support physicians' personal expression, improve camaraderie and improve the dissemination of public health messages. Equally, it risks broadcasting unprofessional content online that reflects poorly on individuals, their affiliated institutions, and the medical profession alike.

For example, let us consider a hypothetical tweet from a female doctor to her colleague describing a recent patient: 'Just saw an 18-year-old unmarried G5P0, with Chlamydia, herpes, and

gonorrhea. Disgusting!’ This tweet would have contravened a few of Wilkinsons (2009) so-called ‘behaviors inherent to good medical professionalism.’ This doctor should have had “respect for her patients’ diversity” and shouldn’t have been so judgmental (in this case, about the patients alleged sexual promiscuity and lifestyle). She also should have upheld patient confidentiality (as although the patient’s name wasn’t tweeted, the descriptors used about her obstetric and sexual histories would surely have made her easily identifiable amongst her friends and family who might have come across this tweet). The doctor should have, in my opinion, had better regard for professional boundaries and exercised greater judgment and discretion.

Defining unprofessionalism online and policing it has been challenging. However, with the increase in awareness of such occurrences, regulatory bodies have published various documents in an attempt to regulate physician’s activities on social media sites. The General Medical Council (GMC) has attempted to do exactly this with its paper. It warns against the blurring of boundaries between ones public and private lives and advices that privacy on these sites cannot be guaranteed. Furthermore, it stresses that physicians must be careful with regards to patient confidentiality, elaborating that although one piece of information may not breach confidentiality by itself, together, a few may certainly do so. In summary, physicians must be cognizant of patient confidentiality and privacy and monitor their Internet presence to ensure that information posted is both

accurate and appropriate. With regards to interaction with patients through social media, again, this interaction should fall within the boundaries of established professional norms. If a physician feels that such an interaction transgresses such norms, he/she should report the matter to the relevant authorities. Finally, it is imperative that physicians realize that inappropriate online interactions may have a negative impact on their reputations and that of their institutions, career advancements, and, perhaps most damning, may serve to undermine public trust in the medical profession as a whole.

**References and Further Reading**, click [here](#).

# Communication and Interpersonal Interactions

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by Vijay Nagpal and Bret A. Nicks

## Introduction

Emergency Medicine and the situations within the department can present a stressful, rapidly changing environment where it may feel as though there is too little time for effective patient communication, patient-centered care or the opportunity to establish an appropriate provider-patient relationship. It is also an environment unlike any other in medicine, where a unique team of individuals faces varying degrees of chaos with limited available information to work together and address the medical conditions of those presenting to the department. Few would recommend entering such an environment in the absence of an established care process and means of clear communication. The tone of the department is set prior to walking into the ED; from the moment you walk into the department, preconceived notions and prejudices remain at the door.

It is no surprise that high-functioning emergency departments have high-performing, well-communicating teams. Clearly defining and communicating to every team member why we are there and how we care for patients sets the tone for every interpersonal interaction. This is true not just for our patient-provider interactions but our interactions with the nursing, ancillary and consultant staff as well (Gluyas, 2015). Establishing a team mentality and acknowledging the value of contributions our colleagues and staff bring to the ED is essential to practicing high-quality, safe emergency medical care. Additionally, the skill set that those in



other health professions bring to the team can help us to look from a different perspective to better understand our patients and facilitate the best care that can be offered in the ED (Klauer & Engel, 2013).

## Essentials of Communication

The approach to providing quality patient care in the ED starts with recognizing the patient-provider mismatched perspective on what has happened and what is occurring (Helman, 2015). It is essential to recognize the patient-physician relationship starts with a significant power imbalance. Attempts should be made to normalize or reduce this power imbalance, to empower the patients and their families. This will enable an open discussion about their medical concerns and assist in making informed decisions about their care. It is important to acknowledge the wait or process they have already endured before seeing you. Thank the patient (and family) for coming to the ED and allowing you to address their medical concerns. Also, take the time to introduce yourself to everyone in the room with the patient and find out who they are in relation to the patient. This can help establish rapport with the patient and those around them (Chan 2012, Cinar 2012, Hobgood 2002).

While many believe the environment of care is the greatest limiting factor as opposed to quality communication, literature would suggest otherwise. Establishing a positive patient-provider relationship is essential for patient care. One must recognize that

while you may not be able to solve the patient's condition or chronic illness, using effective communication skills and providing a positive patient experience will assuage many patient fears (Mole, 2016). Keep in mind, in general, patients remember less than 10% of the content (what was actually said), 38% of how you say it (verbal liking), and 55% of how you look saying it (body language) (Helman, 2015).

## Effective provider communicators routinely employ these 5 Steps

### 1. Be Genuine

We know it. People can sense the disingenuous person – whether it is a gut feeling or through other senses. Try to see the situation from the patient's perspective, and it will ensure that you are acting in his best interest and with integrity.

### 2. Be Present

As emergency providers, we are interrupted more than perhaps any other specialty. However, for the few moments that we are engaged with the patient or his family, be all in. If there is a planned interruption upcoming, make it known prior to starting a discussion. Be focused on them and the conversation; value what they have to share. At the end of your encounter, briefly summarizing what the patient has told you can help to reassure the patient that you were listening and also give them the chance to clarify discrepancies.

### 3. Ask Questions

To effectively communicate, one must listen more than he talks. After introducing yourself, inquire about the patient's medical concern; give them 60 seconds of uninterrupted time. Most patients are amazed and provide unique insights that would otherwise not be obtained. Once the patient has provided you with his concerns, begin asking the specific questions needed to further differentiate the care needed. By asking questions and allowing for answers, you make it about them and give them an avenue to share with you what they are most concerned about, enabling you to address those concerns.

### 4. Build Trust

Given the nature of the patient-provider relationship in emergency medicine, building trust is essential but often difficult. Building trust is like building a fire; it starts with the initial contact and builds with each interaction. Trust is also built on engaging in culturally acceptable interactions (Chan, 2012) such as a handshake, affirming nod, hand-on-shoulder, or engaging posture.

### 5. Communicate Directly

Ensure that at the end of your initial encounter you have established a clear plan of care, what the patient can expect, how long it may take, and when you will return to reassess or provide

additional information. Doing this also allows the patient to be more involved in his care and ask further questions regarding his workup and treatment plan. Additionally, helping the patient to understand what to expect while in the department can help to alleviate fear associated with unannounced tests or imaging studies, especially when these tests may require him or her to be temporarily taken out of the department (e.g., a trip to the CT scanner).

Many of these concepts have been identified in patient satisfaction and operational metrics. In one study, wait times were not associated with the perception of quality of care, but empathy by the provider with the initial interaction was clearly associated (Helman, 2015). In addition, patient dissatisfaction with delays to care is less linked to the actual time spent in the ED and more with a failure to set time expectations about the care process, a perceived lack of personal attention, and a perceived lack of staff communication and concern for the patient's comfort.

### Empathy

In the ED, it is essential to understand that much of a patient's care relates to empathy – the ability to understand and share another person's experiences and emotions. It is recommended to try and understand the patient's agenda. One can accomplish this by asking, "Help me understand what brought you in today." "Help me understand what I can do for you." "Tell me more." This

will help to normalize the patient's situation and gain unique insights into his care concerns.

There are four easy steps to improve reflective listening and perceived empathy in the ED:

1. Echo – Repeat what the patient says; this gives the message that you heard the patient.
2. Paraphrase – Rephrase what the patient says as this gives the message that you understand the patient.
3. Identify the feeling – Say, for example, “you seem frustrated,” “worried,” “upset.” This produces trust.
4. Validation – Validate the patient's feelings verbally by saying statements such as “I can see why you feel that way.”

There is also a great online module and mnemonic for Empathetic Listening skills development (SMACC, 2016). The RELATE mnemonic is:

- Reassure – share your qualifications and experience
- Explain – describe in clear, concise language what the patient can expect
- Listen – not just hearing, encourage the patient to ask questions
- Answer – summarize what they have said and confirm their understanding

- Take Action – discuss and define the care steps (and what to expect)
- Express Appreciation – thank the patient for allowing you to care for them

## The Approach

As with many life circumstances, effective communication is the glue that helps establish connections to others and improve teamwork, decision-making, and problem-solving. It facilitates the ability to communicate even negative or difficult messages without creating conflict or distrust. Recognizing this helps provide the best foundation and approach for successful patient communication, an essential element in the ED. In addition to understanding the five steps of effective communication, ones approach to effective communication must also be guided by the individual patient and adjusted accordingly. So, consider seeing your approach from the patient's perspective, and set the tone with the following three starting points.

## The 3 Starting Points:

### 1. Approach and Appearance:

- Dress appropriately
- Sit down next to the patient
- Maintain an open posture (avoid crossing your arms)

- Maintain good eye contact, if culturally appropriate
- Smile appropriately, nod affirmingly

## 2. How you speak

- Speak slowly and quietly (given the constraints of the ED)
- Use a low tone in your voice
- Empathy can be heard in your tone

## 3. What you say

- Introduce yourself in a culturally appropriate manner
- Use the patient's last name (helps to minimize power imbalance)
- Acknowledge everyone in the room and ask what their relationship to the patient is (i.e., shake hands if culturally appropriate)
- Adjust medical wording based on patient's medical literacy

In addition to understanding the five essentials of communication and setting the tone for the initial care approach, it is important to understand a few of the common reasons communication either fails or succeeds in the Emergency Department. While a single approach framework doesn't always fit, there are some essential **Do's and Don'ts** that must also be considered.

## Do

- Let the patient tell his/her story (Roscoe, 2016)
- Establish what the patient's agenda is, what his/her fears are
- Provide the patient with information regarding what will happen during his/her stay. This puts the patient more at ease and improves satisfaction (Hobgood, 2002).
- Provide expected wait times. Some experts suggest overestimating the time for results and consultant services (Disney Technique).
- Explain the reasons for delays and apologize for it
- After your history and physical, map out the next steps in the process (i.e., establish expectations).

## Don't

- Fold your arms over your chest as this displays an aggressive posture
- Ask why the patient did not come in earlier
- Say, "I guess."
- Repeatedly ask, "why."
- Use the words "never" or "always."

## The Difficult Patient

When facing difficult patients in the emergency department, understanding the situation and the motivation for the patient may help to navigate better the communication challenges that are present. A difficult patient encounter in the emergency department can often be frustrating for both the physician and the patient. These patients often present with chronic medical issues that are superimposed onto individuals with social disparities (Hull & Broquet 2007, Dudzinski & Timberlake 2016). These are just a few examples of types of patients that one may encounter in the emergency department:

### Patient Type and Suggestion

#### *Angry Patient*

Don't ignore the fact that a patient may be angry or upset – often it is related to delays, expectations or care concerns. Try to explore this emotion by asking neutral and non-confrontation questions. Acknowledgment and a simple apology for process issues may prove invaluable.

#### *Manipulative Patient*

While these patients may clearly have a secondary agenda, their medical complaints may still be legitimate. Approach these patients with an open mind, but be prepared to say no to requests that are not clinically indicated.

#### *Frequent Fliers*

High recidivism may be frustrating, but it is important to understand that there may be an underlying reason for frequent ED visits. Socioeconomics and poor access to care are common reasons. Knowing the available resources (e.g., social workers, clinical support nursing) can make a difference.

#### *Combative/Agitated or Intoxicated Patient*

It is most important to keep both the patient and the staff (including yourself) safe. Redirecting the patient and emphasizing the importance of caring for them medically may help to calm the situation. Psychopharmacological intervention may be necessary at times.

For a deeper dive into effective patient communication related to managing difficult patients, **listen** to Episode 51: Effective Patient Communication – Managing Difficult Patients by Anton Helman.

## The Handoff

Communication between providers and patient care transitions present one of the well-known challenges in patient care and errors in care management. This handoff communication, often perceived as the “gray zone,” has been characterized by ambiguity regarding the patient's medical condition, treatment, and disposition (Akper, 2007). Communication errors, particularly related to patient hand-offs, account for nearly 35% of ED-related



care errors. Establishing a standardized process to ensure the quality and clarity of transitions in care are essential. One such example is the I-CAN format that is specifically focused on the ED patient population.

## ED-based Patient Handoff Tool (I-CAN)

### I – Introduction

Briefly describe what brought the patient into the emergency department today. For example, the patient is a 53 yo male with a past medical history of COPD who presents today with a productive cough, wheezing, and shortness of breath.

### C – Critical Content & Interventions Performed

Relate information that helps the receiving provider understand the ED course taken up to this point.

For example: On initial evaluation, the patient was unable to speak in full sentences, and O2 saturation was 88% on room air. We started him on NIPPV, and Nebulizer treatments were given. Respiratory burst steroids have been given to the patient here in the department.

### A – Active Issues

Give the provider an idea of the patient's current condition at this time. For example, the patient improved with an hour of NIPPV and was transitioned to high flow nasal cannula with O2

saturation at 93%. We are currently attempting to wean O2 requirement as tolerated.

### N – Next Steps and Anticipated Disposition

Describe to the receiving provider what will need to be followed up and the anticipated disposition of the patient. For example, the patient will need to be admitted for a COPD exacerbation with a new O2 requirement. He can go to a floor bed if he remains stable on nasal cannula.

While many examples for a unified handoff exist, identifying a defined approach and establishing the expectation for routine use, especially when integrated into the electronic health record at transitions of care, ensure improvement with patient care, quality, and throughput (Akper 2007, Rouke 2016). If the patient and family are involved with this handoff, not only will they understand care expectations but they will also better understand issues with delays, next steps, and care updates.

### Conclusion

Most agree that providing patient care in the ED poses many challenges. The situations we work with can present a stressful, rapid environment where it may feel as though we have too little time for effective patient communication, patient-centered care or opportunity to establish a great patient experience. However, it is also evident that improved communication between the care team and patients improves not only the care experience but also patient care outcomes. Quality communication improves patient

outcomes, compliance, and satisfaction – not to mention the job and team satisfaction. While many techniques exist to improve ED communication, establishing a culture in the ED to habitually adapt these practices is essential. The ED is an environment unlike any other in medicine, where a unique team of individuals works in varying degrees of chaos with limited available information together to address the medical conditions of those presenting to the department. Doing so with effective communication can make a difference.

**References and Further Reading**, click [here](#).

# Data Gathering

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by Chew Keng Sheng

## Introduction

Although a medical student has always been taught to take a comprehensive history and a complete physical examination from head-to-toe, she may find this methodical approach a challenge in the emergency department (ED). Many of the patients who come to the ED are often first-time patients, unfamiliar with procedures, and have diverse complaints ranging from a manipulative attempt to obtain a sick leave certificate to a complex, life-threatening situation. This challenge is further compounded by the fact that many patients in the ED are suffering from acute illnesses or injuries that compromise their cognitive capacity to comprehend and respond.

## The Emergency Medicine Approach

Although some studies have shown that history-taking alone can determine the diagnosis in up to 75-80% of the cases (Hampton 1975, Peterson 1992) obtaining such a comprehensive history in the ED can be an extremely daunting task especially if the patient is extremely ill.

In such a situation, the linear clinical approach – history first, followed by physical examination and investigation – may not be feasible. Rather, data gathering from the patient's history, physical examination, and investigation may need to be performed concurrently. The most important element in the approach to the patient in emergency medicine is to establish the composite initial impression of

the patient. This is based on data gathering from multi-sources including the history, physical findings, and bedside investigations. Of particular importance is answering the vital question: is there any life or limb threat in this patient? And once a life or limb threat is identified, immediate measures must be initiated to reverse the insult before moving on in the data gathering process.

### Activity 1

Watch a [video podcast](#) on General Approach to the Emergency Department Patient.

#### Discuss

- What are the strengths and limitations you see in this emergency medicine approach model where all processes of data gathering (history-taking, physical examination, and investigation) may have to occur simultaneously as compared to the traditional linear clinical approach?

As tough as it may seem, a doctor working in the ED must still establish a good communication rapport with the patient, as much as possible. To attain this, one must utilize open-ended questions.

### Ask the 5-Ws and 1-H questions: “What?” “Why?” “Who?” “When?” “Where?” and “How?”

Pay particular attention to any symptom developed acutely. Acute onset of a headache, for example, suggests a vascular origin. If a patient has had a chronic, persistent or recurrent condition, the important question to ask is “Is there any difference between the symptom before and the symptom now?” A patient with a migraine headache, for example, can present with a sudden “worst ever headache” suggestive of subarachnoid hemorrhage rather than a chronic migraine. If we do not ask for the symptom pattern changes, the patient may not volunteer this information.

#### What

What is the message that the patient is trying to convey to me through the words he does and does NOT use? Observe the non-verbal communication cues that he is trying to convey, e.g., a sense of nervousness, fidgety movements, etc. Often, patients are prone to conceal sensitive information such as sexual history as well as psychiatric/psychological complaints that may only be detected through non-verbal cues.

#### Why

Examples: Why does the patient choose to come in the middle of the night? Why does the patient choose this form of treatment

and not another? Why does the patient think that his or her illness is not serious?

## Who

Examples: Who is/are taking care of the patient at home? Whom does the patient seek advice from when he/she is sick? Who else knows about the patient's illness? Who is/are the eyewitnesses of the accident or the trauma that the patient was involved in? Who is the patient's next of kin? Who can be a legitimate surrogate decision maker for the acutely ill patient?

## When

Examples: When does the pain occur? When does the patient first notice the swelling, the discoloration, etc.? A sudden onset of symptoms is a warning sign and may suggest a vascular event.

## Where

Examples: Where did the accident happen? Where does the patient come from? How far from the hospital?

## How

Examples: How did the accident happen? Did the patient lose his/her consciousness before or after the event?

## Non-verbal cues

Be attentive to the patient's non-verbal cues as well, not just the verbal contents of his visit. Albert Mehrabian, a professor of psychology, developed the classic 7-38-55 rule. This rule consists of the following: while 7% of what the patient communicates comes from the actual words used (the content), 38% of the message comes from the way it is said (the tone), but 55% of the message comes from the non-verbal cues including but not limited to the facial expression, eye contact, etc.

Does the patient appear fearful and defensive? Aggressive? Angry? Disinterested? Click [here](#) to watch a video on Mehrabian's study.

This is especially so when the patient is trying to communicate across sensitive information such as his sexual history or psychological symptoms. Unfortunately, it was found that only between **20 – 40% of doctors** responded positively to the patient's verbal and non-verbal cues (Beckman 1984).

Allow the patient to describe his/her concerns using his own words without interruptions. It has been found that a doctor interrupts his patients as early as **18 seconds** into the conversation, even though it takes at least 150 seconds for the patients to tell his stories (Beckman 1984).



## Activity 2

*Watch* this short [video](#): Presenting your patient to your attending in Emergency Medicine by Dr. David Pierce

*Reflect*: In the video, Dr. Pierce admonishes his residents not to miss anything important by thinking of 5 other differential diagnoses. Why is it especially important to adopt a broad-based approach in diagnoses formulation in the ED?

## Activity 3

*Watch* this [video](#): Approach to the ED Patient.

*Discuss/reflect* on the following questions:

1. In his talk, the speaker stated that “most patients do not take going to the ED casually.” How does knowing that most patients do not take going to the ED casually affect the way you view your patients, especially in the middle of the night?
2. The second thing that the speaker said is that fear and anxiety are routine emotions experienced by ED patients. Do you agree with this statement? If yes, why do you think this is so, and how would this affect your data gathering process? In your ED rotation or posting, observe whether it is indeed true that fear and anxiety are routine emotions experienced by the patients you see. Do you think the doctors have done enough to

alleviate these emotions of fear and anxiety in their clinical encounters?

3. The speaker also talked about the long waiting time in the ED. How does the long waiting time affect your data gathering process?

**References and Further Reading**, click [here](#).

# Diagnostic Testing In Emergency Medicine

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by Yusuf Ali Altunci

## Case 1

*A fifty-one-year-old male patient is admitted to your emergency department (ED) with chest pain that started 30 minutes ago. On his ECG, there are 2 mm ST elevations at DII, DIII, and aVF derivations. Do you need high sensitive troponin analyzes results for acute management of this patient?*

## Case 2

*A thirty-five year- old female patient presented to your ED with sudden onset shortness of breath. She has tachycardia. There is no pathologic finding at auscultation. Her blood pressure is 90/60 mmHg. In history, there is swelling and pain on her left leg for two days. She is using oral contraceptives. For this patient can normal D-dimer result rule out pulmonary embolism?*

## Introduction

The emergency physicians frequently make difficult clinical decisions with limited information while encountered with a multitude of demands and distractions (Kovacs & Croskerry 1999).

EDs are crowded places. Usually, you have limited time to diagnose and treat the patients. Today, diagnostic tools are better than they were in the past. This may help provide an easier diagnostic approach, but the difficulty is knowing how and when you should use these tools. Even if the technology has become available more frequently in clinical practice, clinical expertise and skills are still important factors for making correct, timely diagnoses in patients (Wahner-Roedler 2007).

So this triggers the question: is there one diagnostic approach for each emergency illness that can render the best result for the patient, maximize timeliness and accuracy, and limit cost? This is the essential question that clinical decision

rules try to answer; therefore, the development of these reliable clinical decision rules is imperative for the advancement of modern emergency medicine (Pines 2012).

*“Listen to your patient; he is telling you the diagnosis.” – William Osler (1849-1919)*

## Diagnostic Testing Approach

Polymorbid patients, different diagnostic and therapeutic options, more complex hospital structures, financial incentives, benchmarking, and perceptual and societal changes cause pressure on doctors, especially if medical errors come up. This is especially true for the ED structure, where patients encounter delayed or erroneous initial diagnostic or therapeutic actions and expensive hospital stays due to sub-optimal triage (Schuetza 2015)

Diagnostic tests should primarily be ordered to rule in or out a particular condition based on the differential

diagnosis found through the patient's history and physical examination (Wald 2011). For emergency management, it is usually more important to rule out life threatening pathologies.

So why do we need diagnostic tests? For detecting the problem, of course; however, the decision to test is impacted by multiple factors such as clinical suspicion, persuasion, physician's decision, consultant's or patient's request (Wald 2011).

Patients often express strong preferences for medical tests or treatments of their own choosing, even when physicians believe that those interventions are not beneficial (Brett & McCullough 2012). Patients are also increasingly willing to challenge physicians' intellectual authority, often requesting interventions based on media publicity about new research findings, sometimes before physicians are even made aware of them. Internet sources with clinical information also empower patients to make medical

judgments independent of consultations with physicians (Brett & McCullough 2012). The Internet continues to create new, unschooled Internet doctors and, in turn, new challenges.

Choosing the test or not test in the ED also depends on the resources of the hospital. Some hospitals allow easy access to radiographic testing and laboratory testing. In other hospitals, obtaining a diagnostic test may not be that simple (Pines 2012).

Questions for diagnostic strategy described by Wald (2011) are

- What am I going to do with the test results?
- How is this test going to help me confirm or exclude the diagnosis?
- How will the test result affect my diagnostic strategy, management, or final disposition?

*“Medicine is a science of uncertainty and an art of probability.” – William Osler (1849-1919)*

## Statistics

You decided on one of the diagnostic tests for your patient. Do you think you should know some statistics in order to evaluate the results? Let's check some basic statistical terms that we regularly face as a doctor.

Random ordering of laboratory tests and shortcomings in test performance and interpretation may cause diagnostic errors. Test results may be vague with false positive or false negative results and generate unnecessary harm and costs. Laboratory tests should only be demanded if results have clinical consequences (Schuetza 2015).

**Sensitivity** refers to the likelihood of a test being positive or abnormal in the presence of disease.

•  $\text{Sensitivity} = \frac{\text{True Positive}}{\text{True Positive} + \text{False Negative}}$

**Specificity** refers to the likelihood of the test being negative or normal in the absence of disease

•  $\text{Specificity} = \frac{\text{True Negative}}{\text{True Negative} + \text{False Positive}}$

A test that has high specificity means that it has a low rate of reporting false positives. A test that has low specificity has a high likelihood of false-positive results (Wald 2011).

**Positive predictive value (PPV)** refers to the likelihood of the patient truly having the disease when the test is positive or abnormal.

•  $\text{PPV} = \frac{\text{True Positive}}{\text{True Positive} + \text{False Positive}}$

**Negative predictive value (NPV)** refers to the likelihood that the patient does not have the disease when the test is negative or normal (Wald 2011).

- $NPV = \frac{\text{True Negative}}{\text{True Negative} + \text{False Negative}}$

## Probability

The other important element in testing is the probability. Previously, the physicians' role in emergency medicine was clinical problem solving by history taking and examination only. Now it has changed and incorporates determining the pre- and post-test probabilities essential for the ordering and interpretation of laboratory tests (Schuetza 2015). Probability relates to your concern about a particular patient having an illness or condition and how that concern may or may not be impacted by the diagnostic test results (Wald 2011).

## Testing-related diagnostic error

The EDs are often described as a diagnostic testing center where the results of most diagnostic tests are known within a few hours. The importance of diagnostic tests in Emergency Medicine is an undeniable fact. For example, there are a lot of diagnostic imaging alternatives available in the ED including USG, CT, and MRI in the ED. So, the pathologies that were mostly detected at autopsies in the past, such as pulmonary embolism or an aortic aneurysm, became a clinical problem for today (Wald 2011). Unfortunately, many "routine" laboratory tests are being ordered in "bundles" without any impact on diagnostic or therapeutic management (Schuetza 2015).

Five causes taxonomy of testing-related diagnostic error (Epner 2013)

1. An inappropriate test is ordered.
2. An appropriate test is not ordered.
3. An appropriate test result is applied incorrectly.
4. An appropriate test is ordered, but a delay occurs somewhere in the whole testing process.
5. The result of an appropriately ordered test is not accurate.

## Diagnostic Strategy

Diagnostics, including point of care testing in the ED, is still evolving. As our technology continues to improve, we will have greater access to the results of a multitude of diagnostic studies in a timely fashion (Wald 2011). It is our responsibility to practice medicine in a cost-effective manner that benefits our patients and does not overburden them and the health care system with unnecessary and, at times, overused testing (Wald 2011).

Blood circulating biomarkers play a crucial role in the present diagnostic workup of ED patients. A biomarker may be considered as any protein or other macromolecules that can be objectively measured and evaluated as an indicator of normal biological processes, pathological processes, and course of diseases or pharmacological responses to a therapeutic



intervention. Readily measurable biomarkers give important information about etiology of a disease and the necessity for interventions and prognosis. Diagnostic biomarkers justify the presence or absence of a disease (Schuetza 2015).

In Emergency Medicine practice, we use algorithms or clinical decision rules (Ottawa Ankle Rules, PECARN minor head trauma algorithm, etc.) to make standard management. These are useful and practical tools to make an acceptable decision. Clinical decision rules try to make objective criteria that may help you to distinguish who requires a test or not (Pines 2012). Some people call it “cookbook” medicine, and, of course, “one size cannot fit all.” Today, however, they are the most evidence-based approaches to pathologies. So staying within the rules is one of the best methods that will assist you when contemplating when to utilize diagnostic tests.

Comprehending the evidence behind diagnostic testing and using clinical decision rules to decide when not to test is at the center of emergency medicine practice (Pines 2012).

Last questions that you should keep in mind:

- Will that test result change your management?
- Do you have any plan if it's positive, negative, or indeterminate?

These questions should be considered before you order the test. It is our responsibility that giving the best, correct, and the fastest

management to our patients. However, in the same time, it is our responsibility to use our resources wisely. Therefore, ordering the appropriate tests is very important. The tests which you think it will change your management and you know what are you going to do with the results are the best tests for your patients. In addition, this approach will help to use our resources efficiently and decrease the cost of unnecessary tests.

**References and Further Reading**, click [here](#).

# Creating Your Action Plan

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by Chew Keng Sheng

## Introduction

As the patient's physiologic condition is dynamic and changes from time to time, we need to remember that the action plan is not static and can change in a moment. As such, we must not be too fixated with our earlier impression and fail or refuse to change it in light of discriminating evidence. This is further compounded by the challenge that the emergency department (ED) can be a high-acuity clinical environment that does not afford us the luxury of providing care in a structured manner as a low-acuity outpatient setting does.

Although establishing a definitive diagnosis is the goal in a conventional clinical approach, that is nearly impossible given the limited available clinical and laboratory data as well as the limited time we can spend with the patient in the ED. Unfortunately, establishing the definitive diagnosis may often be an unrealistic expectation of the general public. In fact, some patients are admitted to the hospital, and others are discharged home without a definitive diagnosis. Coming to terms with this unpleasant uncertainty of emergency medicine is necessary. It is, therefore, important to always maintain a healthy degree of skepticism in patient management by asking questions like, "What if I am wrong?" "What else could this presentation be due to?" "Do I have sufficient evidence to support or refute this diagnosis?"

A doctor working in the ED needs to have adequate knowledge of emergency conditions commonly presented to the ED. An emergency condition is any medical condition of sufficient severity (including intense pain) and when the absence of immediate medical attention could reasonably be expected to result in mortality and morbidity. Hence, unlike in conventional patient approaches, working in the ED requires a doctor first to ask this important question, “Is there a life or limb threatening condition that I must rule out in this patient”? A life-threatening condition is a threat to the airway, breathing, and circulation. Once a life or limb threatening condition is identified, interventions must be instituted immediately to address it before moving on to another form of examination and investigation.

## Importance of Vital Signs

In addition to knowing emergency conditions, it is essential not to forget to look at the vital signs chart when formulating your action plan. Bear in mind that “normal” vital signs can be abnormal (Markovchick 2011). For example, an elderly patient with BP that usually ranges from 140 – 160/90 – 100 mmHg can mean that he is unstable with a BP of 110/70 mmHg and persistent vomiting and diarrhea. A patient with severe asthmatic exacerbations who was tachypneic and restless initially does not mean that he is now stable if he is “calmer” with a respiratory rate reduced to 10 breaths per minute. In other words, noting the trend of the vital signs is much more important than reading an isolated vital sign measurement.

## Temperature

Patients in the extreme age group may not mount a sufficient febrile response to an infection to cause an elevation in body temperature. Always remember to ask whether the patient has taken any anti-inflammatory or antipyretic medications (e.g., paracetamol, aspirin, non-steroidal anti-inflammatory drugs) before coming to the ED. The thermoregulatory center is located in the anterior hypothalamus; thus, any central nervous system infection or injury that affects the hypothalamus such as cerebrovascular accident and subarachnoid hemorrhage may affect thermoregulation. Certain drugs (e.g., anxiolytics, antidepressants, oral antihyperglycemics, beta-blockers), adrenal insufficiency, end-stage renal disease and thyroid disorders can also affect basal body temperature or temperature regulation.

## Pulse

When taking the pulse, the rate, regularity, and volume should be noted. The pulse rate should also be interpreted taking into consideration the patient’s age. For adolescents and adults, the maximum sustained HR estimation can be calculated with the this formula: **maximum sustained HR = (220 – age in years) × 0.85.**

Bradycardia is defined as a heart rate lower than 60 beats/min in adults. However, a well-conditioned athlete may have a normal resting heart rate as low as 30 to 40 beats/min. Ask also if the patient is taking any medication that could affect the pulse rate.

For example, digitalis compounds,  $\beta$ -blockers, and antidysrhythmics may alter the normal heart rate and the ability of this vital sign to respond to a new physiologic stress.

Physiologically, for every one-degree increase in Fahrenheit, the heart rate increases by ten beats/min. As 1 Celsius equals to 9/5 or 1.8 Fahrenheit, the increase of every one-degree Celsius results in an increase of pulse rate by 18 beats/min. This is known as the Leibermeister's rule. However, there are conditions whereby the increase in temperature is not followed by an increased pulse rate. This is known as relative bradycardia (or the Faget sign). Causes of relative bradycardia can be divided into infective and non-infective causes. Infective causes include the following: Legionella, Psittacosis, Typhoid Fever, Typhus, Babesiosis, Malaria, Leptospirosis, Yellow fever, Dengue fever, Viral hemorrhagic fevers, Rocky Mountain spotted fever, etc. The non-infective causes beta-blockers (but not an angiotensin-converting-enzyme inhibitor, ACE inhibitor; calcium-channel blocker nor digoxin), central nervous system lesions (tumors and bleeds), lymphomas and drug fever (Cunha 2000).

## Respiratory Rate

The respiratory rate only informs us how fast or slow the breathing rate is; it does not inform us about the depth of the breathing or the oxygenation status of the patient. Therefore, besides looking at the rate, we should also pay attention to the

depth of breathing and the pulse oximetry for the oxygen saturation.

Respiratory rate of >60 breaths per min in an acutely ill child under the age of 2 months is a predictor of hypoxia. Respiratory rate generally increases in the presence of fever; therefore, it can be difficult to determine whether the tachypnea is a primary finding of respiratory problems or is simply associated with the fever itself. Observe the breathing patterns of the patient as well. Look for any abnormal breathing patterns such as Cheyne-Stokes breathing (episodes of progressive shallow-deep-shallow cycles suggestive of stroke, trauma, carbon monoxide poisoning, and metabolic encephalopathy, etc.) and Kussmaul breathing (increased rate and depth of breathing). Click here for a video of [Cheyne-Stokes breathing](#) and a video of [Kussmaul breathing](#).

## Pulse oximetry

Pulse oximetry is a non-invasive measurement of the oxygen saturation. The relationship between SaO<sub>2</sub> and the partial pressure of arterial oxygen (PaO<sub>2</sub>) is described by the oxyhemoglobin dissociation curve (ODC). Because of the sigmoid shape of the ODC, a unit reduction of PaO<sub>2</sub> change in this relatively flat portion of the ODC produces only a small change in SaO<sub>2</sub> as compared to a unit of reduction of PaO<sub>2</sub> in the relatively steep part of the curve that produces a much greater degree of reduction of PaO<sub>2</sub>. The point of intersection between the relatively flat portion of the curve and the relatively steep portion

of the curve is known as the ICU point, and it corresponds to a SaO<sub>2</sub> of around 92% and the PaO<sub>2</sub> of 60 mmHg. Therefore, always attempt to maintain the SaO<sub>2</sub> above 92%. PaO<sub>2</sub> below 60 mmHg means that the patient can markedly desaturate. Conversely, at a PaO<sub>2</sub> above 60 mmHg, increasing the PaO<sub>2</sub> will not result in a marked increase in the SaO<sub>2</sub>. In fact, giving too much supplemental oxygen may result in an ever increasing PaO<sub>2</sub> with a SaO<sub>2</sub> maintained at 100%. Hyperoxia (too high PaO<sub>2</sub>) can be harmful as it can lead to adverse effects such as generation of reactive oxygen species and release of angiotensin II resulting in vasoconstriction. (Click [here](#) to access two articles for more explanation and diagrams: Hooley J. [Decoding the Oxyhemoglobin Dissociation Curve](#) and Brandis, K. [Oxygen Dissociation Curve](#)).

## Blood pressure

Blood pressure, defined as the force exerted by blood on the vessel wall, only indirectly measures perfusion, as blood flow equals to the change in pressure divided by resistance. But because peripheral vascular resistance varies, normal blood pressure does not necessarily mean good tissue perfusion. The normal blood pressure may be “maintained” by an increase in peripheral vascular resistance. Furthermore, hypotension is a late sign of shock; this is especially true in children. For example, in class II hemorrhagic shock (with a loss of 15%–30% blood volume), the findings usually include tachycardia, tachypnea,

cool, clammy skin, and delayed capillary refill. However, the systolic blood pressure (BP) is still within the normal range even though the pulse pressure is decreased. The decrease in pulse pressure is due to the increased levels of circulating catecholamines, causing an increase in peripheral vascular resistance, and raising the diastolic BP.

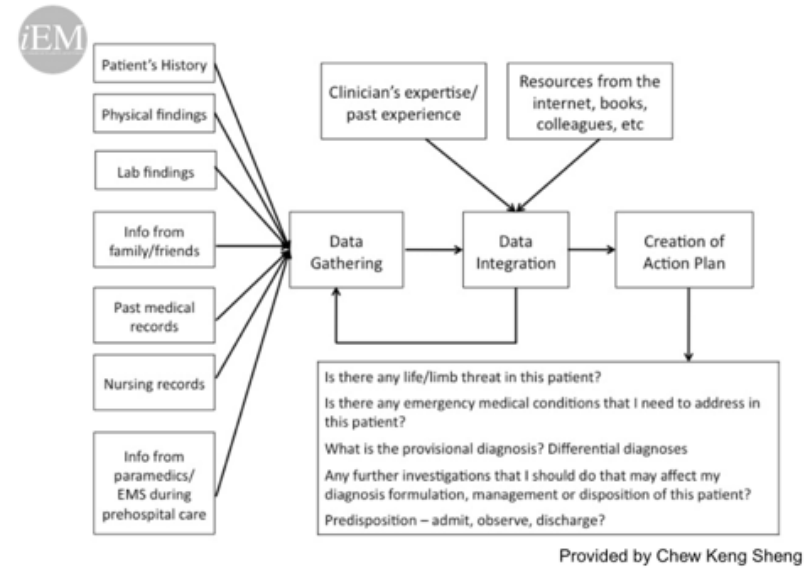
For children, the blood pressure measurement varies according to age. A formula for estimating the 95th percentile BP (normal) in young children is as follows: **BP = 80 + (2 x age in years)**. Hypotension is defined as less than the 5th percentile BP that can be estimated by the following formula: **hypotension = less than 70 + (2 x age in years)**.

The algorithm of [data gathering](#) and creation of an action plan in the ED is shown below.

**References and Further Reading**, click [here](#).



**Diagram 2.1** Data gathering and creation of action plan



# Documentation

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by Muneer Al Marzouqi and Qais Abuagla

## Introduction

Whether you are rotating in the Emergency Department (ED) or elsewhere, one of the key skills to learn is how to write a complete and legible patient record. Documentation in the ED is usually challenging, as it may be difficult to adequately capture and note details down in a timely manner. This happens especially when dealing with high acuity or critical case scenarios. Even as a medical student or intern, your medical record is important on so many levels. It serves to reflect your general approach, thought process, the care you provided to patients, as well as potentially identifying gaps in your knowledge and training. Attendings, clerkship directors, and faculty usually emphasize and pay attention to how notes are written and

may use them for summative or formative assessments as well as a means for feedback. These documents are also an important tool for communication between the ED and respective physicians, specialties and other stakeholders. Appropriate medical documentation improves the quality of communication within an ED and aids in the quality assurance process.

*It is said that “if something was not written in the chart, then it never happened.”*

Having a well-organized and legible chart gives the auditors and reviewers a clear picture of the physician’s thought process, the actions he/she performed, and provides a real-time

snapshot of the patient's general condition at any given encounter. There is always room to learn about and improve medical documentation. Therefore, this section will review the key elements used when documenting in the ED (Murphy, 2001; CDEM, 2010)

## Emergency Medicine Note

Before writing your note, the nursing triage notes and vital signs, if available, need to be reviewed. If obvious discrepancies are seen, these need to be verified with the nurse and patient, as there may be errors. In addition, any abnormal vitals in triage must be acknowledged and written in the note.

Like any other medical record, the ED document will comprise of the patient's history, physical exam findings, differential diagnoses, investigations ordered, lab and imaging findings, assessment and plan. Each component will be discussed separately, and suitable examples will be provided accordingly (CDEM, 2010; Carrol, 2016a and 2016b).

## History

When writing the patient's history, one needs to be clear, thorough, and concise avoiding any long and complex phrases. Ideally, it needs to flow in a logical and chronological sequence. Unnecessary details are better avoided as they serve as distractors and may confuse other readers. Recording the date and time when the patient was seen is crucial, especially in critical patients, as it helps create a timeline for when time-

sensitive interventions were done or when medications were administered (Carrol, 2016a and 2016b).

## Components of the history include

### 1. Chief Complaint

This usually includes the presenting complaint, ideally in the patient's words, with the duration (Example: Abdominal Pain – for two days).

### 2. History of Present Illness

In general, there are two formats when writing a history of present illness (HPI), the narrative format and bullet points format. Both are acceptable as long as the history is written in a comprehensive, concise and coherent manner. It is of added value if pertinent negatives and positives are added when writing the HPI, to show the physician's thought process. This will lead the person reading the chart towards what differential diagnoses to consider and what to rule out, depending on what the patient is presenting with. Certain mnemonics may be used to aid in writing a systematic HPI (Example: OLDCARS or OPQRST).

**Example 1:** A 45-year-old man, with a history of Coronary Artery Disease and Hypertension, presenting to the ED with chest pain that started 3 hours ago. The pain was of gradual onset while sitting on his chair, localized in the center of the chest and lasted

for 20 minutes. It was described as “a heavy boulder on my chest.” The pain started when he had a quarrel with his daughter and was relieved with sublingual nitroglycerin. It was associated with nausea and sweating, but no vomiting. Was localized and not radiating to the shoulders or arms. He claims it was moderately intense at 4/10 on the pain scale. He denies any shortness of breath, palpitations, dizziness, or abdominal pain.

**Example 2:** A 26-year-old male, previously healthy, presents with a sore throat for one week. It is associated with subjective fever and fatigue. It is aggravated whenever he drinks or eats but denies any difficulty swallowing or drooling. The patient also denies any chills, runny nose, cough, night sweats, or shortness of breath. No recent travel history reported. Has several sick contacts at home with similar symptoms.

### 3. Review of Systems

Other organ systems and symptoms that were not mentioned in the HPI are to be reviewed to make sure the patient does not have other complaints or organ system involvement. If the review or system (ROS) cannot be obtained because of the patient’s underlying condition (i.e., unconscious, critically ill, or having dementia), this should be noted in the chart. Generally, ask patients questions from head to toe (Example: “Do you have a fever, chills, headache, sore throat, chest pain, abdominal pain, urinary symptoms, etc.”). Document all positive ROS symptoms and state the remaining ones as otherwise normal.

### 4. Past Medical/Surgical History, Medications, and Allergies

List any known active illnesses the patient might have or had in the past. Include any surgical procedures he had. State what medications he is actively on and whether he has any drug or food allergies.

### 5. Family and Social History

Document a brief family history that may be relevant to the chief complaint (Example: Family history of Diabetes and Cardiac Disease in a patient presenting with chest pain). Social history mainly includes asking about smoking habits, alcohol consumption, sexual history and illicit drug use. It also might be important and relevant to ask about the patient’s financial and health insurance status, particularly in certain healthcare settings, to avoid ordering unnecessary tests and paying extra costs.

## Physical Examination

When recording physical examination (PE) findings start with the patient’s general appearance and vital signs, highlighting abnormal ones. It is important not to document or fabricate any findings that were not examined; committing to such findings may have medical and medico-legal implications that are best avoided. Document all findings from examined systems including findings from inspection, palpation, auscultation, etc. There is no need to document findings that are not pertinent to the chief complaint (Example: Neurological examination findings in a

patient with a sore throat). Include important positive and negative findings for any given case (Carrol, 2016a).

Example: Patient with abdominal pain

- Important positive findings: Soft, non-tender abdomen, normal active bowel sounds
- Important negative findings: No rebound tenderness, guarding, rigidity, or peritoneal signs

## Assessment

Should capture the essence of the case and defend the rationale for potential further investigations. It usually includes an objective summary of the case with differential diagnoses based on history and physical examination findings.

## Plan

This section includes what investigations, medications, procedures, and consultations are to be ordered or performed. Time of consultation is very important, and the doctor's name and his/her recommendations are to be documented in a timely manner.

## Disposition

This usually is the last part of the note. It mentions whether the patient is going to be admitted, discharged, or transferred to another facility. If discharged, follow-up instructions and return

instructions should be documented clearly (CDEM, 2010; Carrol, 2016a and 2016b).

## Summary of all components in an ED Note:

1. Chief complaint
2. History of present illness with pertinent positives and negatives
3. The brief review of systems
4. Focused past medical and surgical history
5. Focused pertinent medications and allergies
6. Very focused family and social history if required
7. Vital signs, highlighting any abnormal readings
8. Focused and pertinent physical exam
9. Assessment
10. Plan
11. Disposition

## Few helpful hints during documentation

- Place a date and time for all notes in the medical record
- Write notes clearly and legibly



- If you make a mistake, draw one line through it and sign your initials
- Document a focused but thorough History and Physical Examination
- Avoid using unclear abbreviations that are not used commonly
- Document vital signs and address abnormalities
- Document the results of all diagnostic tests that were ordered when appropriate
- When speaking to a consulting service, document the name of the physician and the time the call was made
- Document the patient's response to therapy
- Document repeat examinations
- Document your thought process (medical decision-making)
- Avoid writing derogatory comments in the medical record
- Avoid changing or adding comments to the medical record after completion. It may be appropriate to add an addendum but only if it is properly timed and dated.
- Document all procedures performed

- If a patient leaves against medical advice (AMA), document that you have explained the specific risks of leaving AMA to the patient and relatives
- Document plan for outpatient care and follow-up
- If using an electronic medical record (EMR) instead of a handwritten one, all of the above sections, components and hints apply (Murphy, 2001; Dunbar, 2014; Virtual Mentor, 2011)

Sample ED Note, please click [here](#).

**References and Further Reading**, click [here](#).

# Discharge Communications

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by Justin Brooten and Bret Nicks

## Introduction

The process of patient discharge from the emergency department (ED) provides critical information for patients to manage the next steps of their care. Hospital accreditation and governmental organizations often require these instructions for quality or monitoring metrics. However, studies show that many patients do not fully understand or recall the instructions they receive (Clarke, 2005; Clark, 2005). Add to this the myriad challenges inherent in every emergency department that only perhaps compound this lack of comprehension and subsequently impact care compliance, outcomes, and patient experience.

In many situations, the discharge process is often limited to a brief exchange of documents, prescriptions and verbal description of the diagnosis, frequently leaving patients with uncertainty about their care plan. Certainly, understanding discharge instructions can be very challenging. At the time of discharge, patients or family members may be experiencing physical and emotional discomfort. They may be eager to leave, and thus, less interested in the instructions. Moreover, a significant number of patients have low literacy or health literacy levels (Zeng-Treitler & Hunder, 2008). Also, the busy ED setting may distract the patient's attention from such instructions. Therefore, understanding the challenges around discharge communications in the ED from the patient's perspective and having a clear approach and purpose is essential. Discharge is not an afterthought; it is the

first step of a patient's care transition and greatly impacts quality outcomes, litigation, experience and team morale (Henry, 2013; Siff, 2011).

## Understanding the Challenges

Emergency physicians face unique challenges while ensuring high-quality care due to distractions and time limitations that are common throughout ED settings. In most cases, emergency physicians have little or no previous knowledge of their patients, making effective communication paramount when patients are discharged from the ED (Jon, 2013). Recognizing the value of early quality communication continued throughout the patient care encounter may carry over to the discharge care processes and, in turn, improve an important aspect of quality and patient-centered emergency medical care.

It has been demonstrated that many patients are discharged from the ED with an incomplete understanding of the information needed to care safely for themselves at home (Clarke, 2005; Crane, 1997; Engel, 2012; Sameuls-Kalow, 2015; Taylor & Cameron, 2000). Patients have demonstrated particular difficulty in comprehending post-ED care instructions regarding medications, home care, and follow-up expectations. And while all patients discharged from the ED should be provided instructions for ongoing management of their medical condition, studies have demonstrated that the patient recall and understanding of diagnosis, treatment, and follow-up plan are

quite poor (Clarke, 2005; Clark, 2005; Crane, 1997; Engel, 2012; Sameuls-Kalow, 2015; Taylor, 2000; Zeng-Treitler & Hunder, 2008). This raises significant concerns for care plan adherence and medical outcomes. Given current trends toward value-based care and the fact that nearly half of the lawsuits in emergency medicine revolve around discharge instructions and the discharge program given to patients, ongoing improvements in the discharge communication process is essential (Henry, 2013; Siff, 2011). While some of this relates heavily to the ability of the provider to establish a trusting and positive patient-provider relationship within the ED constraints, several strategies can be used to enhance the recall of instructions, improve compliance, and minimize litigation.

## Discharge Essentials

Effective discharge communication provides an opportunity for the ED team to summarize a patient's visit, teach them how to care safely for themselves at home and provide specifics regarding the next steps in their care process. It also gives ED physicians a chance to address any remaining questions or concerns (Jon, 2013), often augmenting patient and family understanding while improving care plan retention. Although patient education at discharge typically begins with initial assessments and conversations with the patient and his family, other factors can also influence the success or failure of how information is transmitted at discharge (Jon, 2013).

Common interventions included in an effective ED discharge process consist of a standardized approach (content), information delivery, confirmation of comprehension, post-discharge care follow-up planning, review of vital signs and a patient-centered closure (Table 2.1) (Taylor, 2000; Zeng-Treitler & Hunder, 2008).

**Table 2.1** Table 1. Interventions in the ED Discharge Process

DOMAIN	INTERVENTION
Content	Standardize approach
Delivery	Verbal instructions (language and culture appropriate) Written instructions (literary levels) Basic Instructions (including return precautions) Media, visual cues or adjuncts
Comprehension	Confirm comprehension (teach-back method)
Implementation	Resource connections (Rx, appointment, durable medical supplies, follow-up) Medication review

Content refers to the education provided to our patients related to the treatments, tests, and procedures performed during the ED visit, as well as further education on diagnosis, treatment plan, the expected course of illness and medication reconciliation. It should also include time-sensitive and specific information associated with their diagnosis and care plan regarding what to do and when to do it. This should include precautions about when to return to the ED versus waiting for any follow-up appointments, and what steps have already been taken to assist with this process. Utmost clarity regarding what type of follow-up

is needed and why, as well as how to care for oneself until that time, improves outcomes and compliance. Some have phrased these basic tenants of discharge as the ‘rules of the road’; however, this may serve as the basis from which to develop your process.

## Rules for the Road

1. Have the right diagnosis
2. Time & Action Specific Instructions
  - What to do
  - When to do it
3. Provider Specific
  - Who to contact
  - Why and When
4. Printed Information, Verbally Explained
  - Verbally confirmed

The quality and approach of a physician’s delivery of content cannot be overstated. The ED provider and care team members must consider the wide range of literacy (and health literacy), cultural backgrounds and access to outpatient resources when delivering the ongoing care instructions (Engel, 2012; Sameuls-Kalow, 2015). In many instances, to improve patient

understanding of discharge instructions, EDs attempt to improve patient and family understanding of discharge instructions through standardization and simplification of written and verbal instructions for patients and those with them. This verbal discussion can be especially helpful for those with low health literacy. Also, utilizing interpreter services for those who speak other languages may be vital. Other approaches that may benefit patient outcomes include providing supplemental written information and using visual and multimedia adjuncts to support understanding (Taylor & Cameron, 2000; Zeng-Treitler & Kim, 2008). Essential to any successful approach is the patient's comprehension of the information provided. After all, if the content and delivery are exceptional but the comprehension is poor, this should be seen as a discharge failure as it decreases care compliance and outcome quality. To address this specific aspect of the discharge process, instituting a read-back or teach-back method is recommended.

The implementation of discharge care processes frequently falls short due to unidentified social and medical factors that prevent the plan from being carried out. Social factors could include homelessness, low income, uninsured/underinsured status, lack of transportation, or lack of primary care. Medical factors could include concurrent psychiatric illness, substance abuse, cognitive impairment, inability to care for self, or young/advancing age. Understanding these circumstances will help identify patients at

high risk of discharge failure and trigger additional resource considerations for these patients.

The discharge process provides an opportunity to ensure the patient's condition is well understood that there aren't any additional medical red flags that need to be addressed, and that the care plan and follow-up are fully comprehended. In an online video, Dr. Oller (2016) provides another process to engage the 'moment of safety' related to discharge and outlines five essential steps for any ED discharge.

## **ED Discharge: Moment of Safety**

1. Has the medical provider discussed the findings, diagnosis, and plan of care (including medications and follow-up plan)?
2. Confirm the discharge instructions and prescriptions match the patient identifiers
3. Review all prescriptions and clarify any changes
4. Review of current vital signs
5. Closure

Watch the [video](#).

## **Barriers to Successful Discharge**

The barriers to successful discharge are myriad. Some are intrinsic to the ED work environment and the nature of ED patient arrival and flow. Others relate to the challenging or often



unidentified social and medical factors that prevent the plan from being completely carried out. In a recent American College of Emergency Physician Quality Improvement and Patient Safety Section meeting, Dr. Pham (2016) shared a conceptual framework for understanding the barriers to success and improving the discharge process (Figure).

While this framework may not be uniformly representative of all EDs, it addresses many of the operational failures that occur outside of the ED and outlines opportunities for hospitals and health systems to align with improved patient care outcomes. Moreover, while screening for high-risk discharges in EDs occur, the additional resources needed to ensure appropriate social work or case management care coordination are often limited.

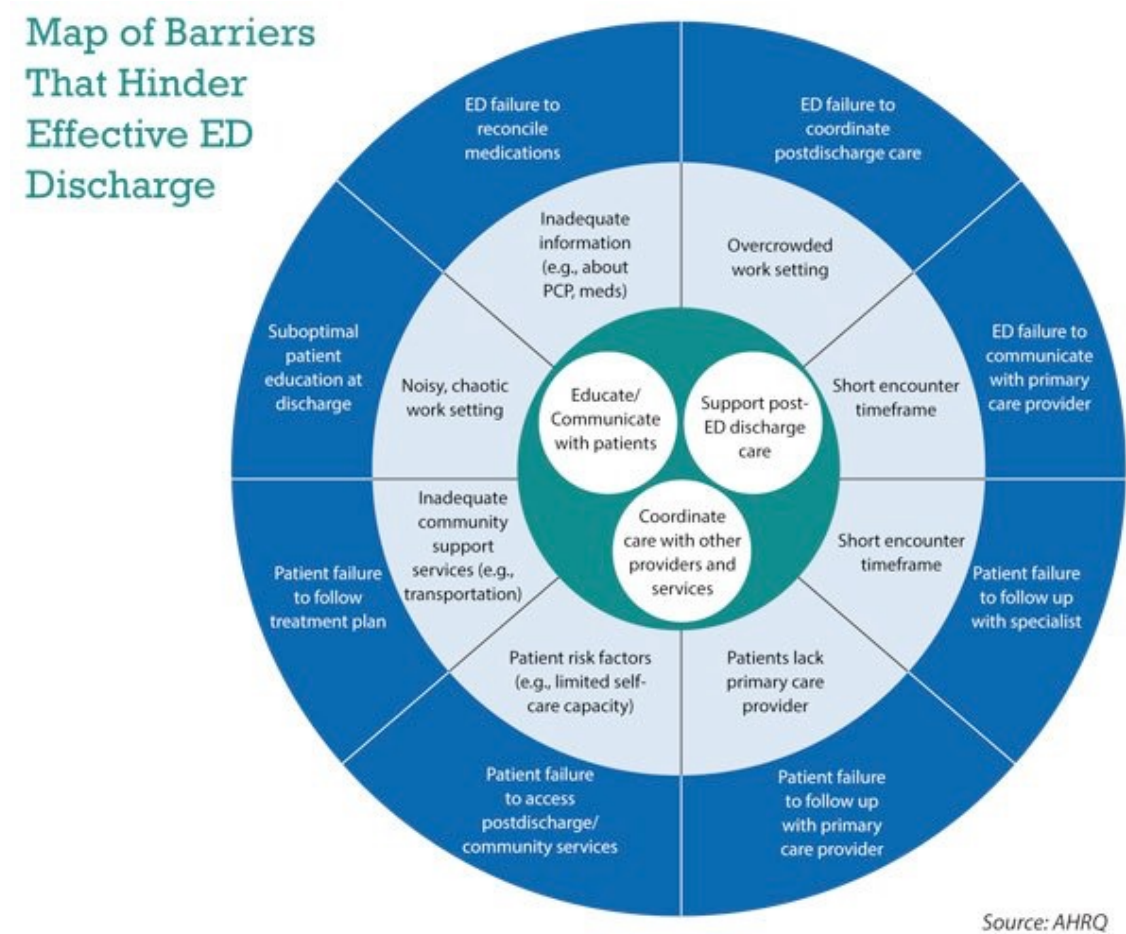
Post-discharge follow-up processes for patients at risk for failing discharge instructions exist in some systems. This may include flagging a patient’s chart for a social work follow-up to assess and assist with the patient’s ability to obtain necessary medications, obtain follow-up appointments, or address other concerns identified by the provider. Some physician groups routinely call the patient the next day to see how the patient is doing and ensure understanding of his/her discharge instructions and care plan (Sameuls-Kalow, 2015; Taylor 2000).

### Types of Discharge Information Packets

Discharge instructions vary widely by practice location and resources available. However, there remain three primary means

of providing discharge information and instruction: basic care instruction note, a pre-formatted illness specific instruction sheet, and templated software-based discharge product (Taylor, 2000).

**Diagram 2.2** Barriers of effective ED discharge



Commonly used, an instruction note is simply a set of instructions handwritten or typed on plain paper, without the assistance of computer programs. In settings with limited resources, this may be the only means of providing essential care information for the

patient, their families and the provider with whom they may follow-up. While uniquely tailored, they may lack substantial content for care, take time to prepare, and are limited by literacy and handwriting.

Information sheets are pre-printed education and instruction documents that describe care information related to one specific illness. They can be developed for the most common medical illnesses for each institution and have essential information regarding plans of care. Information sheets are immediately available, inexpensive, reproducible, and can be designed to include simple language and or pictorial education. They are not patient-specific, may not provide adequate instruction in difficult or complicated cases and require computer, printer, and copying capacity.

For settings with an integrated EHR, software products that create discharge packets (including discharge diagnoses, medications, medical care instructions and information regarding the illness, outlined care course after leaving the ED and essential contact information for those next steps) are available. These are highly resource dependent and, therefore, may not be routinely available.

## **Against Medical Advice and Elopement**

In certain circumstances, patients may request to leave prior to completion of their medical evaluation and treatment. In this situation, it is essential for the last health care professional caring

for the patient to document clearly why the patient left and attested that the patient had the mental capacity to make such a decision at that time (Henry, 2013). While some electronic documentation systems have templates in place to assist with this documentation, Table 2.2 provides basic information for against medical advice (AMA) discharge documentation that can be used to create a uniform template (Henry, 2013; Siff, 2011; Levy, 2012; Devitt, 2000).

An attempt should be made to provide the patient with appropriate discharge instructions, even if a complete diagnosis may not yet be determined. Include advice for the patient to follow up with his physician, strict return precautions, and concerning symptoms that should prompt the patient to seek further care. It should also be made clear that leaving against medical advice does not prevent the patient from returning to the emergency department for further evaluation if his symptoms worsen, or if he changes his mind. Despite a common notion to the contrary, simply leaving against medical advice does not automatically imply that physicians are immune to potential medical liability (Levy, 2012; Devitt, 2000). If a patient lacks decision-making capacity to be able to adequately understand the rationale and consequences of leaving AMA and his condition places him at risk for imminent harm, involuntary hospitalization is warranted. In unclear circumstances and if available, psychiatry can assist in determining capacity, especially in the case of patients with mental health conditions.

Elopement is a similar process where patients disappear during the care process. While it is difficult to provide discharge paperwork for these patients, documenting the actions taken to find the patient is essential (e.g., searching the ED, having security check the surrounding areas). In addition, attempt to reach the patient by phone to discuss his elopement and any additional care issues or concerns. Documentation of these attempts or any additional conversation is very important (Henry, 2013; Siff, 2011).

verbal instructions remain more effective than written instructions, but both are needed. Be explicit, keep it simple and have the patients repeat back instructions to ensure understanding. These simple steps will improve patient outcomes, compliance and avoid legal pitfalls.

**References and Further Reading**, click [here](#).

**Table 2.2** Documentation for Patients Leaving Against Medical Advice

COMPONENT	DESCRIPTION
Capacity	Establish patient's decision-making capacity, and clarify aspects of care which may affect capacity (i.e. Patient is now clinically sober, etc.)
Risks	Specific condition associated risks that were discussed (missed diagnosis, potential harms from untreated disease process, etc.)
Verify comprehension	Patient's understanding of the risks
Patient's decision	Include patient's decision, and any alternative plans (i.e. patient refused admission, but agreed to follow up with primary physician tomorrow.)
Signatures	Patient's and provider's signatures

## Conclusion

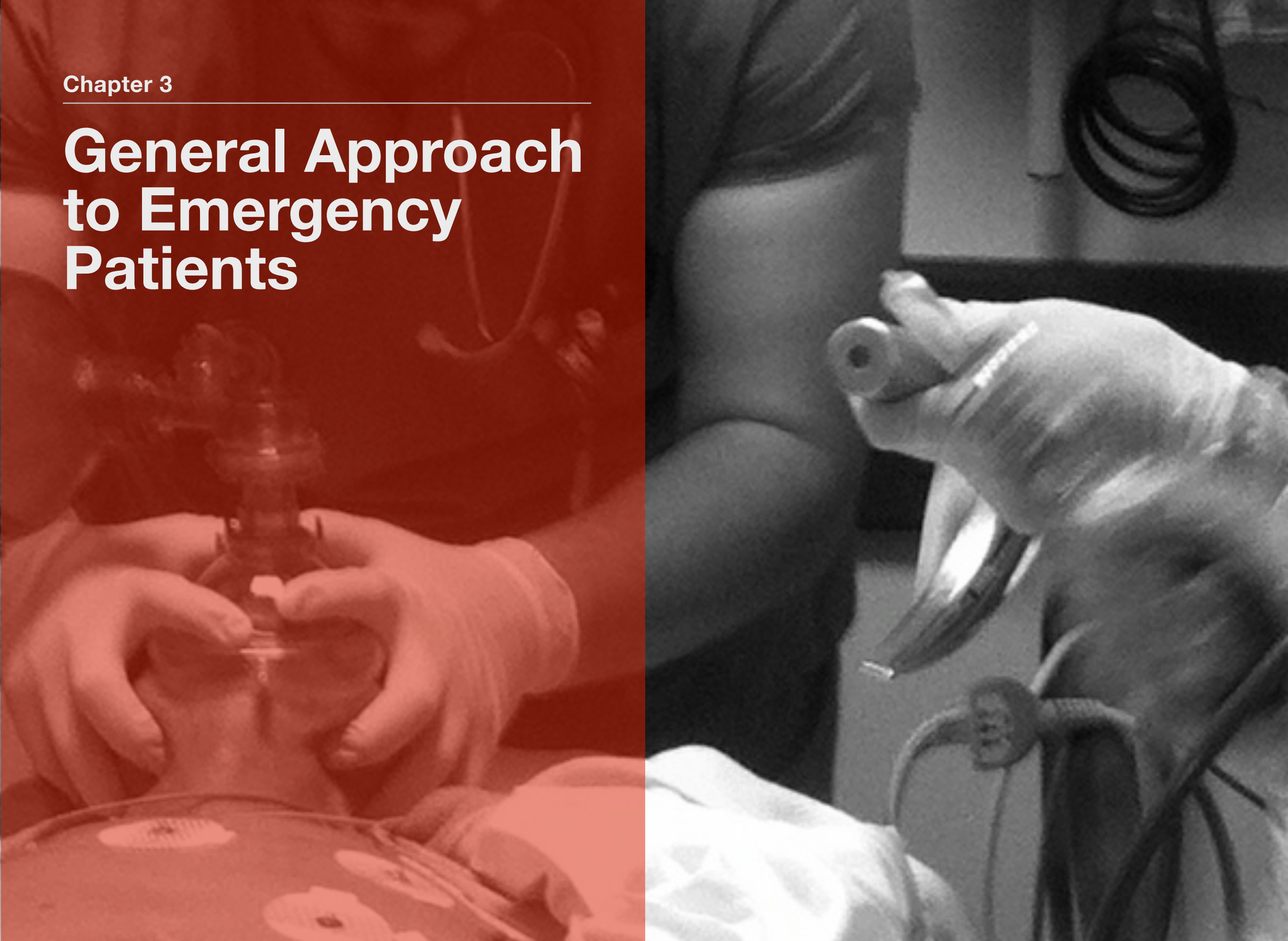
Discharge instructions are a very important part of the ED care process and record. It is essential to ensure each patient has a complete understanding of her instructions and to recognize that



## Chapter 3

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# General Approach to Emergency Patients



# The ABC Approach to the Critically Ill Patient

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by Donna Venezia

## Introduction

### History of the ABC's

The basic ABC algorithm was initially designed and implemented on a large scale in the early 1960's for those requiring cardiac-pulmonary resuscitation. The order has recently been changed to the CAB for those who have suffered a cardiac arrest (See BLS/ACLS – **Cardiac Arrest section**). Twenty years later the American College of Surgeons again modified this sequence for patients with acute traumatic injuries. Over the subsequent years, there have been many variations and modifications for the variety of critically ill patients presenting to an emergency department. Most recently, the addition of point-of-care testing with ultrasound

has been incorporated into algorithms further to refine the accuracy of initial treatment in the critically ill.

### Goals of approaching any critically ill patients are

- Rapidly identify and manage life/brain-threatening conditions before the exact diagnosis is made.
- After initial stabilization, follow with full history, exam, time-consuming lab/radiological testing and reach the final diagnosis.

## Identifying A Critically Ill Patient

Triage is a reliable method to quickly select from a large group of waiting patients, those who may have a potential illness requiring time-critical management to save a life or the brain.



As a standard structure, currently, all modern emergency departments have a triage unit to prioritize the patients. It aims to select more critical patients as early as possible and create an appropriate patient flow in the emergency department. However, triage can be done in the field by EMS staff, and patients may directly bring to the resuscitation room.

### Potential critically ill patients may present with:

- altered mental status (unresponsive or confused/agitated)
- noisy respiration (gurgling, stidor, wheezing)
- inability to speak normally (acute hoarseness or inability to articulate words)
- respiratory distress (rapid/deep or slow/shallow/agonal respirations)
- acute weakness or inability to ambulate (diffuse/focal muscle weakness or light-headedness/syncope)

- acute torso discomfort (may be associated with radiation to jaw, anterior neck or shoulder/medial upper arms) suggestive of an MI/ cardiovascular problem.
- severe acute headache
- intractable seizure (may not show muscular signs after a period of time)
- history of significant trauma, drug ingestion, exposure, suicidal/homicidal ideation
- significant vital signs abnormalities (age-dependent)

### Point of Care Testing

- adjunct tests/equipment that help guide early decision-making
- results should be back within seconds to minutes, not hours!

### The ABCDEF Sequence

- Each letter represents a crucial body system that, if significantly disrupted and left untreated over hours rather

than minutes, can result in death or brain damage.

- The order is performed sequentially to avoid skipping crucial steps and generally to manage the most serious first, however, the sequence can and should be performed simultaneously (horizontal approach) in those with multiple life-threatening conditions if there are enough team members. Modify as appropriate to the individual.
- Because management may need to be simultaneous, the team approach is crucial in successfully resuscitating any critically ill patient.
- It is also important to emphasize that the availability of various treatment modalities at each medical facility.

### Meaning of the letters in the ABCDEF sequence:

A = Airway Disorders with C-spine control

B = Breathing Disorders

C = Circulation/Cardiovascular Disorders

D = Disability (Neurological Disorders)

E = Exposure/Decontamination

F = Fever (Extreme Temperature Disorders)

### How to approach the critically ill patient using the ABCDEF algorithm

For each letter or body system:

- obtain a brief, focused history and exam
- obtain available point-of-care testing to aid in the evaluation/management
- initiate management for any acute life or brain threatening condition
- then, proceed to the next letter and repeat
- if no intervention is needed, quickly proceed through the sequence. (Evaluation of a normal person should take just a few minutes or even seconds.)

## A – Airway with C-spine Control

### Focused clinical assessment for impending/actual airway compromise:

- noisy respirations (gurgling, stridor, choking sounds) with or without retractions
- drooling, inability to swallow secretions, leaning forward in a tripod position
- throat swelling sensation with or without pain
- change in voice associated with symptoms of bacterial infection or allergy (hoarseness, “hot potato” voice)
- active retching or vomiting with an inability to turn or move to protect from aspiration
- oral exposure to fire/steam inhalation, chemicals, acids/alkali
- neck trauma with crepitus over larynx or expanding hematoma

### Point of Care Testing

- generally, none required for complete obstruction
- soft tissue neck X-ray (for potential/partial airway obstruction only)
- indirect laryngoscopy (for potential/partial airway obstruction only)
- fiberoptic laryngoscopy (for potential/partial airway obstruction only)
- ultrasound (for identification of cricothyroid membrane or assessment proper endotracheal tube placement)

### Emergency Equipment for Managing Airway Problems

1. nasal or oral airway devices
2. suction devices (rigid tip and/or small flexible tip)
3. intubation equipment (i.e., laryngoscopes with ET tubes of various sizes)

4. airway adjuncts for difficult airways – i.e. LMA, iGel, Bougie, video laryngoscope, fiberoptic laryngoscope, etc.
5. Magill Forceps for foreign body removal
6. Pre-intubation supplies – supplemental wall and/or tank oxygen, RSI medication, sedation medication, oral numbing medication
7. “failed airway” kit – cricothyroidotomy kit with appropriate sized Shiley or endotracheal tubes
8. capnography and/or ultrasound to assess for proper endotracheal tube placement
9. appropriately sized cervical collars

### **Management Algorithm for Critical Airway Problems**

- possible c-spine injury – employ the second person to immobilize c-spine.

Only jaw thrust maneuver is allowed in this situation (see caveat 1)

- Tongue obstructs airway in an obtunded patient – perform either head tilt, chin lift, or use jaw thrust maneuvers if possible. See BLS/ACLS.
- obtunded, without trauma – position patient on the side to avoid tongue obstruction
- Patient unable to be positioned – place nasal or oral airway. Avoid oral airway if partially awake since may cause gagging/vomiting. Avoid nasal airway if midface trauma.
- pharyngeal secretions, blood, and/or vomitus – suction
- obstructing foreign body – perform abdominal thrusts/chest compressions per BLS or if visible, attempt to retrieve with McGill forceps.
- laryngeal edema; likely anaphylaxis – administer IV/IM Epinephrine, likely

hereditary angioedema – administer C1 esterase compound.

- signs of imminent or complete airway obstruction, unrelieved from above – attempt intubation with the most appropriate device by the most experienced provider. May attempt BVM ventilation first, especially in children with epiglottitis, as a temporizing measure.
- unable to intubate or BVM – immediately perform cricothyroidotomy; avoid if laryngeal fracture a concern – prefer fiberoptic intubation or tracheostomy in OR if possible. (See cricothyroidotomy technique)

### **Caveats**

1. The airway is always associated with the phrase, “with c-spine control”. Before performing any airway procedures, one must quickly assess the likelihood of a c-spine injury. If there is a possibility of an injury in an unresponsive patient, i.e. found at the

bottom of the stairs, or on the side of the road, unconscious, then assume an injury and protect the c-spine by simply immobilizing as best possible. Typically a C-collar is slid under the back of the neck while someone immobilizes the head. If airway management is required, the front of the collar can be opened or removed, as needed, while someone stabilizes the head in relation to the torso. Nothing further needs to be done in the primary survey to evaluate the c-spine.

2. An unresponsive patient has a potential for airway compromise and subsequent aspiration. However, since proper intubation is time intensive, you may avoid intubation in these patients until the primary sequence is completed, unless the patient is actively retching. Have someone prepare the equipment as the sequence is being completed and continue the evaluation since treatment of a condition found later in

the sequence may improve the mental status, making intubation unnecessary such as low blood sugar. Be prepared to log roll quickly if the patient vomits.

### **Conditions causing airway compromise**

- unresponsive patient with tongue blocking the airway an unresponsive patient who is unable to protect from aspiration of blood/vomit, etc.
- an unresponsive patient who is unable to protect from aspiration of blood/vomit, etc.
- infections, i.e. epiglottitis, retropharyngeal abscess, etc.
- allergic reactions/anaphylaxis, airway burns, i.e. steam, chemicals, alkali/acids, etc.
- airway burns, i.e. steam, chemicals, alkali/acids, etc.
- other causes of edema, i.e. ACE inhibitors, hereditary angioneurotic

edema, laryngeal cartilage fractures secondary to trauma

- laryngeal cartilage fractures secondary to trauma
- expanding paratracheal hematoma
- tracheomalacia
- pharyngeal malignancies

## **B – Breathing Disorders**

### **Focused clinical assessment for evidence respiratory failure**

**Cyanosis**, inability to speak full sentences without needing a breath, confused/agitated or unresponsive with:

1. Rate: too slow, shallow, agonal, gasping (age-dependent, generally rates <10 in an adult are abnormal)
2. Rate: too fast and/or deep (again age-dependent but >20 in a resting adult is abnormal, and > 30 is significantly abnormal)
3. Abnormal lung sounds:

- unilateral decreased breath sounds (either dull or hyper-resonant)
- wheezing or poor air movement
- rales (fine crepitation) or rhonchi

#### 4. Chest wall abnormalities affecting breathing dynamics – flail chest/open punctures

Obtain as much focused history/exam as able to help define the need for a particular emergent treatment strategy for the common causes of critical respiratory conditions. For example, two common causes of severe respiratory distress are pulmonary edema and COPD. Both may present with wheezing (“cardiac asthma” in CHF), pedal edema and/or JVD, making the decision for which type of emergent management strategy difficult. Obtain as much focused history/exam in a brief period of time, i.e. family states heavy smoker with similar episodes in the past, all resolved with inhaler therapy or the patient has a history of recent ECHO

with very poor ejection fraction, etc. to help make a decision about treatment.

If still not clear as to a management strategy, add point-of-care testing, i.e., lung sonography or upright portable CXR.

#### Point-of-Care testing

- pulse Oximeter
- CO2 waveform monitor
- arterial (ABG) or venous (VBG) blood gas
- portable CXR (upright, if possible)
- pulmonary ultrasonography:
  - evaluate lung sliding for pneumothorax
  - assess costophrenic angles for effusion/hemothorax
  - assess lung field segments for A/B lines, signs of consolidation

#### Management Algorithm for Acute Respiratory Disorders

•fix all upper airway critical issues first

- slow, agonal respirations or significant respiratory acidosis on ABG – provide BVM ventilation and administer Narcan.
- rapid breathing with hypoxia – provide supplemental O2 by the non-rebreather mask to keep O2 saturation greater than 94%.
- sucking chest wound – seal with an occlusive dressing (3 sides only)
- tension pneumothorax – place a 14 gauge needle, immediately followed by a chest tube
- massive hemothorax/pleural effusion – drain fluid, contact trauma surgeon since may need transfusion/transfer to OR for massive hemothorax
- no improvement in oxygenation despite placement of non-rebreather mask or above procedures, either –



• allow the patient to breathe spontaneously under tightly held BVM mask with PEEP valve on exhalation port and 15 L/min nasal cannula O<sub>2</sub> placed under the mask or

• provide NIV (non-invasive ventilation) with CPAP/BiPAP

- patient agitated and unable to tolerate masks – administer IV Ketamine.
- Signs of obstructive pulmonary disease (COPD/asthma) – administer inhalational beta agonist. Consider additional therapy (i.e., ipratropium, Prednisone, Magnesium, epinephrine, etc.).
- Signs of acute pulmonary edema with adequate BP – administer repetitive or continuous doses of Nitroglycerin SL, spray or IV. Consider additional drug therapy (i.e. Furosemide, etc.)
- **respiratory distress** unresponsive to above therapy – intubate and mechanically ventilate

## Emergency Equipment for Managing Breathing Emergencies

1. Noninvasive ventilator NIV
2. BVM (bag-valve-mask) with O<sub>2</sub> supply and added PEEP valve
3. additional wall or tank for an additional source of O<sub>2</sub> (nasal cannula)
4. 14 gauge catheter-over-the-needle
5. various sized chest tubes

## Caveats

1. fast RR with clear lungs may be secondary to psychogenic hyperventilation, primary brain lesions, metabolic acidosis, poisoning, sepsis, pulmonary embolus or pericardial tamponade:
  - a. consider paper bag breathing if O<sub>2</sub> sats normal, no acidosis, and hyperventilation syndrome most likely (i.e., anxious with carpopedal spasm).

b. Consider specific **poisoning antidotes**, i.e., cyanide antidote or hyperbaric/100% O<sub>2</sub> for CO poisonings. See the **toxicology section**.

c. **Sepsis**, **Pulmonary embolus**, and **pericardial tamponade** management are discussed in more depth in the circulatory section since the primary critical presentation is usually circulatory collapse.

d. Obtain **ABG/VBG** if metabolic acidosis likely, manage most likely cause. See acid/base section.

2. Severe hypoxia unresponsive to therapy, particularly with clear lungs, may be due to shunting from congenital heart disease which, in a neonate, may respond to the administration of PGE<sub>1</sub> (prostaglandin).
3. It is important to recognize that oxygenation and ventilation are different. A patient may not be hypoxic, especially if given supplemental O<sub>2</sub> but may still be in

acute ventilatory failure. (Only 250 cc of oxygen is used by the resting adult per minute. However, 6-10 L of air must be moved per minute to adequately ventilate a normal adult and prevent the rise in pCO<sub>2</sub>.)

### Conditions Associated with Respiratory Failure

- Pulmonary edema
- COPD/asthma
- severe pneumonia
- ALI/ARDS from any cause (drugs, aspiration, etc.)
- tension pneumothorax
- chest wall dysfunction, (flail chest, muscular weakness, open sucking wound)
- respiratory depressants (narcotic OD, sedative OD)
- bronchiolitis

- pulmonary embolus, air/amniotic fluid/fat embolus
- massive hemothorax or massive pleural effusion
- exhaustion from prolonged hyperventilation
- chronic lung conditions: cancer, sarcoidosis, fibrosis, etc.

### C – Circulation Disorders

Poor perfusion, Hypertensive crisis, Acute MI

#### Clinically assess for poor perfusion associated with

- tachycardia: > 100 abnormal in adults, > 150 frequently clinically symptomatic.
- bradycardia: < 60 abnormal, < 30 frequently clinically symptomatic.
- hypotension: systolic < 90
- Perfusion and cardiovascular assessment may include

• Skin – i.e., cool, diaphoresis, pale, poor capillary refill, hives, erythema

• Mental status changes – i.e., confusion, slow responses, agitation

• Rhythm/quality of pulses in all four extremities

• Assessment for hidden blood loss, i.e., rectal for melena, pelvic instability, pulsatile abdominal mass

• history: internal/external bleeding/trauma, vomiting/diarrhea, oral intake/urine output, fever, diabetes/renal insufficiency/cardiac failure, medications, drug abuse/OD, last menses

#### Clinically assess for hypertension associated with

- signs of end-organ damage/involvement, i.e., encephalopathy and/or papilledema, pulmonary edema, cardiac ischemia, renal impairment, and/or neurological abnormalities

- pregnancy (generally 3rd trimester/first weeks postpartum); any new elevation of BP >140/90, particularly associated with a headache, abdominal pain, jaundice, shortness of breath and/or visual disturbances

### **Clinically assess torso discomfort for likely MI**

- description varies; besides chest discomfort, symptoms may include either/or epigastric discomfort, mid-back discomfort, radiation to shoulders, anterior neck, jaw or upper, inner arms.
- Note: There are many serious causes of torso pain, i.e., simple pneumothorax, cholecystitis, pancreatitis, bowel perforation, etc.; work-up and management would be performed during the secondary evaluation unless there are signs or symptoms of circulatory collapse. The evaluation of torso discomfort in the initial assessment should include an emergent EKG to pick up an MI.

### **Point-of-care testing**

- EKG (perform within 10 minutes of ED presentation; may include right-sided leads RV3,4 and posterior leads V8, V9)
- Cardiovascular ultrasound to include assessment of:
  - LV cardiac contractility – normal, hyperactive, weak
  - the ratio of right to left ventricle size
  - pericardial fluid/tamponade physiology
  - aortic root dilation/dissection flap
  - IVC collapsibility with inspiration
  - evidence of free intraperitoneal fluid
  - evidence of abdominal aortic aneurysm and/or dissection
  - evidence of DVT in femoral veins
- Telemetry monitor strip
- ABG/VBG with electrolytes

- Hemocult paper (only needed if any question of blood/melena in stools)
- Urine beta-HCG for critical childbearing age females

### **Emergency Equipment for Managing Cardiovascular Emergencies**

1. pelvic binders/gauze for compression/tourniquets
2. defibrillator/external pacemaker
3. large bore IV's and 0.9% saline or Ringer lactate fluids
4. various sized IO insertion kits
5. central line kits (only for large bore sheath placement, if necessary)
6. thrombolytics or ability to access PCI (percutaneous coronary intervention) facility
7. immediate access to O negative blood
8. straight catheter/Foley catheter (for pregnancy check) and monitoring urine output

## 9. Sengstaken-Blakemore tube

### Management Algorithm for Critical Cardiovascular Disorders

#### • Management of Poor Perfusion

👤 place two large bore IV's and attach telemetry monitor to all (may collect various labs including blood cultures, but should send type and crossmatch now)

👤 evidence of external bleeding, unstable pelvis – apply pressure/binder; in rare cases tourniquet

👤 patient in the 3rd trimester of pregnancy – displace uterus to left/wedge under right flank unable to start IV – attempt IO (intraosseous) with 300 mmHg pressure cuff over the fluid bag to increase flow rate (Central line sheaths, if unable to start IO).

👤 unable to start IV – attempt IO (intraosseous) with 300 mmHg pressure cuff over the fluid bag to increase flow

rate (Central line sheaths, if unable to start IO).

👤 if no evidence of cardiac failure – administer bolus 10-20cc/kg 0.9% NS/ Ringers solution. (Further fluid administration determined by clinical/sono evaluation, risk/benefit, i.e., permissive hypotension and clinical response, i.e., urine output).

👤 Evidence of unstoppable internal bleeding – immediate consultation with appropriate specialty, i.e., surgery, OB, GI, etc. Consider various meds to attenuate bleeding, i.e., Tranexamic Acid, Terlipressin for esophageal bleed or Oxytocin for OB bleed. Consider various procedures to stop internal bleeding, i.e., Sengstaken-Blakemore tube placement for esophageal bleed, uterine massage post-delivery, etc.

👤 severe blood loss and/or persistent unstoppable bleeding – transfuse O-negative units until type specific or fully cross-matched blood available

👤 unstable tachydysrhythmia (not sinus, multifocal atrial tachycardia, junctional) – cardiovert per ACLS

👤 unstable bradydysrhythmia – administer meds (i.e., Atropine, etc.)/ place external pacemaker per ACLS

👤 evidence of rhythm disorder is associated with K<sup>+</sup> abnormality by the lab, clinical history (i.e., renal failure, DM) and/or EKG findings – administer appropriate hyper/hypokalemia/magnesemia therapy

👤 dysrhythmia unresponsive, with evidence of thyroid storm or history of drug OD, consider thyroid management or specific antidotes: NaHCO<sub>2</sub> for fast Na<sup>+</sup> channel OD (TCA, tricyclic antidepressants), Digibind for Digoxin toxicity, etc.

👤 evidence of aortic dissection by clinical, sono evaluation – administer b-blocker first, i.e., Esmolol, then antihypertensive, i.e., Nitroprusside, contact cardiovascular surgery

• evidence of obstructive shock by clinical/sono – treat appropriately as guided by diagnosis, i.e., thrombolytics/interventional radiology for pulmonary embolus, pericardiocentesis for tamponade, chest tube for tension pneumothorax, etc.

• no evidence of acute volume loss and/or no response to fluids or previous therapies – start pressors, Norepinephrine, Dopamine, Epinephrine, Dobutamine, etc., i.e., Epi for anaphylaxis, Dob/Norepi for cardiogenic shock, Norepi for sepsis, etc.

• if no response to above, consider either:

1. adrenal crisis – start IV Hydrocortisone. (Dexamethasone, if choose to perform testing concurrently.) and/or

2. drug OD (i.e., b-blocker or calcium channel blocker – treat with high dose Insulin/glucose)

### **Management Algorithm for Severe Hypertension associated with**

- evidence of end-organ damage (ischemia, heart failure, encephalopathy, etc.) – administer IV antihypertensive (Labetalol, Nitroprusside, etc.) Avoid pure beta blockers if suspect cocaine overdose.
- evidence of hemorrhagic stroke, thrombotic stroke, subarachnoid hemorrhage (See Disability Section)
- pregnancy and new elevation of BP >140/90 – re-evaluate in 15 minutes
- pregnancy with evidence of pre-eclampsia/eclampsia (i.e., headache, nausea/vomiting, abdominal pain, visual disturbances, shortness of breath, hyperreflexia, seizures – with or without proteinuria) – or severe hypertension BP 160/110 – administer MgSO<sub>4</sub> and initiate antihypertensive, (i.e.,

Hydralazine, Labetalol, or Nifedipine), immediate OB consult.

### **Management Algorithm for Torso Discomfort**

- acute torso discomfort with MI documented by EKG – contact cardiology for immediate PCI/transfer and/or administer thrombolytics depending on location and timing of event per ACLS
- acute MI by EKG (whether eligible or not for PCI/thrombolytics) – monitor for dysrhythmia, i.e., ventricular fibrillation, etc., administer Aspirin, follow protocols per local cardiologist.
- high suspicion of cardiac ischemia but EKG not diagnostic – repeat in 10-15 minutes.

### **Causes of critical cardiovascular conditions**

- dehydration
- acute blood loss (internal and external)



- sepsis
- drug toxicity/OD
- cardiogenic shock
- anaphylaxis
- neurogenic shock
- adrenal crisis
- thyroid storm
- obstructive shock
  - pulmonary embolus
  - pericardial tamponade
  - tension pneumothorax
  - gravid uterus compressing IVC
- tachydysrhythmias/bradysrhythmias with or without electrolyte disorders
- symptomatic hypertensive with or without pregnancy
- acute MI
- acute aortic dissection/rupture

## D – Disability (Neurological/Psychological Disorders)

### Clinically assess for

- depressed consciousness (lethargic, confused, comatose) (may use GCS to assess the degree of unresponsiveness)
- pupil size, symmetry, and reactivity
- agitation, delirium (waxing and waning level of consciousness associated with confusion/disorientation and/or hallucinations – typically, visual/tactile)
- acute focal weakness/paralysis, or inability to speak
- severe, acute headache, nuchal rigidity
- signs of status epilepticus, including subtle seizure-like activity (i.e., twitching eyelids, stiffness, persistent unresponsiveness after obvious seizure-like activity)
- acute psychiatric disorder with either suicidal or homicidal ideation

### Point-of-Care Adjuncts

- fingerstick glucose measurement
- non-contrast head CT to be performed in less than 30 minutes
- acute malaria screen in appropriate environments
- rapid HIV test
- electrolytes (Na<sup>+</sup>, and Ca<sup>++</sup>, in particular), if available, on ABG/VBG assessment, sono for evaluation of papilledema
- sono for evaluation of papilledema

### Emergency Equipment Needed for Neurological Management

1. CT scanner
2. access or ability to transfer to neurosurgical equipped OR
3. LP tray
4. leather restraints

5. stretchers that allow for head elevation

## Management Algorithm for Critical Neurological Disorders

### *Acute Agitation/Delirium Algorithm*

- in all patients attempt to talk first to calm and remove anything that might cause injury
- agitation, particularly in young patients or possible drug toxicity/withdrawal – administer Benzodiazepines. Avoid in elderly with dementia; likely to increase confusion. Monitor respirations in all.
- agitation, with signs of hypoxia, hypoperfusion – consider Ketamine starting dose 1mg/kg with continued ABC resuscitation
- agitation, with a known history of psychiatric disorder or likely new-onset psychiatric disease – administer psychotropic agent, i.e., Haldol IV, IM. with or without Benzodiazepine.

- agitation, unable to calm with above and/or patient an imminent danger to self/others – call for ‘man-power’ support and apply four-point restraints. (Provide close monitoring of the patient and remove restraints as soon as deemed safe)

### *Acute Mental Status Depression Algorithm*

- fix the airway, breathing and circulation conditions first
- Check fingerstick glucose – if low administer bolus or drip of D50/D25 or D10 depending on patient age. May give IM Glucagon if unable to start IV and patient cannot swallow. Administer Thiamine with the glucose. (Narcan should have already been given under section B).
- if GCS < 9 after ABC resuscitation – the patient likely requires intubation to protect from aspiration – prepare equipment

•History acute fever, headache, without focal neurological signs, recent seizure history or impaired immunity and exam/sono shows no papilledema – check malaria smear, rapid HIV test, perform LP, initiate empiric antibiotic treatment (possible steroids first), based on age/likely etiology. Before any meds given attempt to quickly determine if allergic, from family, old records, etc.

- History acute fever, headache, with focal neurological signs or seizures, impaired immunity and/or exam/sono shows papilledema – do not perform immediate LP – check malaria smear, rapid HIV test, initiate empiric antibiotic treatment (possible steroids first), based on age/likely etiology. Before any meds given, attempt to determine if allergic, from family, old records, etc. Follow with CT and possible LP, ASAP.
- consider status epilepticus in all non-responsive patients, (motor signs may be minimal) or if not awakening between seizures:

• check electrolytes – if hyponatremic administer 2cc/kg over 10 min of 3% NaCl (max 100cc)

• Third trimester/post delivery – administer MgSO<sub>4</sub>/consult OB

• likely INH OD or neonatal dependency – administer Pyridoxine.

• all others – start with Benzodiazepines, consult neurology

- if no improvement with above – obtain head CT; follow management in the section below.

*Focal Neurological Signs/AMS (with or without head trauma) and/or a Sudden, Severe Headache Algorithm*

- obtain a **head CT** in all patients, if available
- normal CT, likely SAH by history (onset > 6 hours), perform LP – nontraumatic

blood/ xanthochromia, immediate neurosurgery consultation, control BP < 160/90. See SAH guidelines.

- Normal CT, likely thrombotic stroke – initiate TPA/endovascular therapy per protocols, control BP to <185/110. If unable to use TPA, do not drop BP unless >220/120. See thrombotic stroke guidelines.
- New intra-cerebral bleed on CT – control BP to <140/90; reverse anticoagulants. See hemorrhagic stroke guidelines.
- epidural/subdural/nontraumatic SAH on CT – immediate neurosurgery consultation for possible OR/IR intervention.
- Evidence of acute herniation – raise the head of bed 30-45 degrees (assuming no spine trauma), consider Mannitol, 3% NS, and/or mild, brief hyperventilation. Consider IV dexamethasone for a tumor with herniation.

## Causes of critical neurological disorders

- conditions affecting airway, breathing and/or circulation
- metabolic disorders:

• Hypoglycemia/hyperosmolar coma/DKA

• thyroid disorders

• electrolyte disorders (primarily Na<sup>+</sup> and Ca<sup>++</sup>)

• liver/kidney failure, etc.

- drug toxicity/OD or drug withdrawal syndromes
- acute psychiatric disorders
- mass lesions (hemorrhage, tumors, abscesses)
- infections – meningitis/encephalitis (bacterial, fungal, viral, parasitic infections including cerebral malaria)
- status epilepticus and post-ictal states

- stroke syndromes – thrombotic, intracerebral hemorrhagic, SAH

## E – Exposure

### Clinically evaluate

- areas hidden by clothing/body position for missed lesions (rashes/stab/gunshot wounds) by undressing and log rolling.
- the body for evidence of self/child/elder/domestic abuse and evidence of IV drug abuse.
- for possible contaminated clothing/skin: substances absorbed through the skin (i.e., hydrocarbon pesticides), caustics, radiation or objects causing continued burns, etc.

### Point-of-care testing

- none

### Management Algorithm for Exposure Disorders

- signs of child or self-abuse – provide safe location and separate from abusers

- evidence of hidden bleeding – manage as per Section C
- evidence of clothes/skin contamination – decontaminate, according to toxicity and protect self and others in the process (self-protection should be implemented at the onset of patient evaluation)
- re-dress patient in a gown to prevent cooling and provide privacy

### Equipment Needed for Exposure/Decontamination

1. shower with containment for water runoff
2. protective gowns, masks, gloves for staff
3. isolation room with air vent containment
4. shears/metal cutter

## F – Fever (Extreme Temperature Disorders)

### Clinically Assess

- skin warmth/coolness
- skin color (pale/red), dryness, diaphoresis
- muscle rigidity, shivering
- thyroid for nodules/enlargement
- obtain the history of medications (recent psychotropic/succinylcholine, anesthetics, etc.), drug abuse, endocrine disease, outdoor exposure, excessive exercise
- Note: normal temperature is 98.6 F or 37 C. Any temperature above 100.4 F or 38 C rectally is considered a fever. However, it is the extremes of temperature that require emergent management, usually > 105 F (40.5 C) or < 95 F (35 C)

## Point-of-care testing

- Thermometer: oral -affected by mouth breathing, drinking warm/cold fluids
- axillary – add a point to correlate with rectal temperature.
- rectal – most accurately reflects core temperature.

## Emergency Equipment Required

- fan
- ice packs
- lavage tubes
- warming blankets
- rectal temperature probe

## Management Algorithm for Critical Temperature Extremes

### *Hyperthermia Algorithm*

- initiate heat loss for all by

- convection (evaporation) methods, i.e., tepid water spray on skin and fan and/or
- conduction heat loss by placing ices packs over major vessels, i.e., groin, axilla or neck. (Ice tub immersion possible, but not able to easily monitor.)
- cool IV fluids
- if severe temperature elevations – initiate core cooling: ice water lavage of the bladder, thorax, stomach and prevent shivering with drugs such as dexmedetomidine / Butorphanol.
- evidence of anticholinergic, sympathomimetic, MAOI poisoning – consider antidotes.
- evidence of neuroleptic malignant syndrome – stop neuroleptics, consider various antidotes See management of neuroleptic malignant syndrome.
- evidence for thyroid storm – initiate b-blockade, cortisone, PTU, then iodine last

### *Hypothermia Algorithm*

- mild to moderate temperature decline – 30-35 degrees – external rewarm, i.e., blankets and initiate warmed IV fluids, and heated inspired air heated to 45 degrees
- severe, <30 degrees – consider additional core rewarming, i.e., peritoneal lavage, thoracic lavage, esophageal tubes, etc.
- evidence of myxedema coma – administer thyroxine and hydrocortisone, avoid rapid rewarming

## Causes of critical heat/cold related conditions

- exposure to extreme environmental temperature conditions
- endocrine disorders (especially hyper/hypothyroidism)
- toxins/OD's (anticholinergics, sympathomimetic, MAOI drugs, ASA, etc.)



- sepsis (for both extremes)
- neuroleptic malignant syndrome
- malignant hyperthermia associated with anesthetics

After the sequence is completed, quickly re-evaluate the patient to see if intervention(s) resulted in improvement.

Then follow the ABC's with:

- evaluation of past medical history, medication history, and allergy history, if not already performed
- perform the secondary survey (i.e., detailed history and a complete exam)

**References and Further Reading**, click [here](#).

# Abdominal Pain

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by Shaza Karrar

## Case Presentation

*A 39-year-old female presented to the emergency department (ED) complaining of right-lower-quadrant (RLQ) pain; pain duration was for 1-day, associated with nausea, vomiting, and loose motions. Abdominal pain started centrally and was described as diffuse and colic, 3 hours later it gradually shifted to the RL and became continuous in nature. Her Last-menstrual- period (LMP) finished a week ago. She denied any regular medications, known allergies or using any contraceptive pills. Also, she denied any past surgical history, travel history, or eating outside. Upon examination, she was found to be afebrile and vitally stable. The abdomen was soft, non-distended, with RLQ Tenderness, positive rebound tenderness, and positive bowel sounds.*



Audio is available [here](#)

# General Approach and Critical Bedside Actions

## General Approach and Key Concepts

Abdominal pain is a common complaint presented in the ED. It can range from benign, undifferentiated conditions to life-threatening ones. The role of the Emergency Physician (EP) is to identify life-threatening conditions for timely interventions. Life-threatening conditions can stem from various systemic pathologies (i.e. Cardiac, Gastrointestinal GI, Vascular, Urologic, Gynecologic, Infectious, etc.). As well, the EP has to assess properly and dispose of the less serious pathologies; which, if done poorly, can propagate and lead to delayed presentations and higher morbidities (i.e., Gastroesophageal reflux disease GERD, peptic ulcer disease, etc.). Special attention should be given to certain subgroups of patients including the elderly, the immunocompromised, females of childbearing age and children.

## Types of Abdominal Pain

Abdominal pain can be either visceral, parietal or referred in origin (Table 3.1). In certain abdominal pathologies, abdominal pain can typically begin as visceral, dull and diffuse, and then progress to parietal, sharp and localizable, as in Appendicitis. Understanding the basic physiology of abdominal pain can be of immense aid in your assessment and approach.

**Table 3.1** Types of abdominal pain

iEM Table 1: Types of Abdominal Pain			
Origin	Clinical Presentation	Pathophysiology	Clinical Examples
Visceral Pain	<ul style="list-style-type: none"> <li>Vague</li> <li>Dull</li> <li>Poorly localizable</li> </ul>	<ul style="list-style-type: none"> <li>Inflammation, ischemia or distention.</li> <li>Pain can start diffuse/midline stemming from the embryological bilateral innervation of organs.</li> </ul>	<ul style="list-style-type: none"> <li>Early stage of appendicitis, with periumbilical pain</li> </ul>
Parietal Pain	<ul style="list-style-type: none"> <li>Sharp</li> <li>Localizable</li> </ul>	<ul style="list-style-type: none"> <li>Parietal peritoneum inflammation or irritation.</li> <li>In a case of complete involvement of the parietal peritoneum, pain can become more diffuse in advanced disease.</li> </ul>	<ul style="list-style-type: none"> <li>Late stages of appendicitis with localizable RLQ pain and rebound tenderness; due to peritoneal irritation.</li> </ul>
Referred Pain	<ul style="list-style-type: none"> <li>Distant to original pathology</li> </ul>	<ul style="list-style-type: none"> <li>Related usually to embryological origins.</li> </ul>	<ul style="list-style-type: none"> <li>Myocardial Infarction (MI) presenting as epigastric pain.</li> </ul>

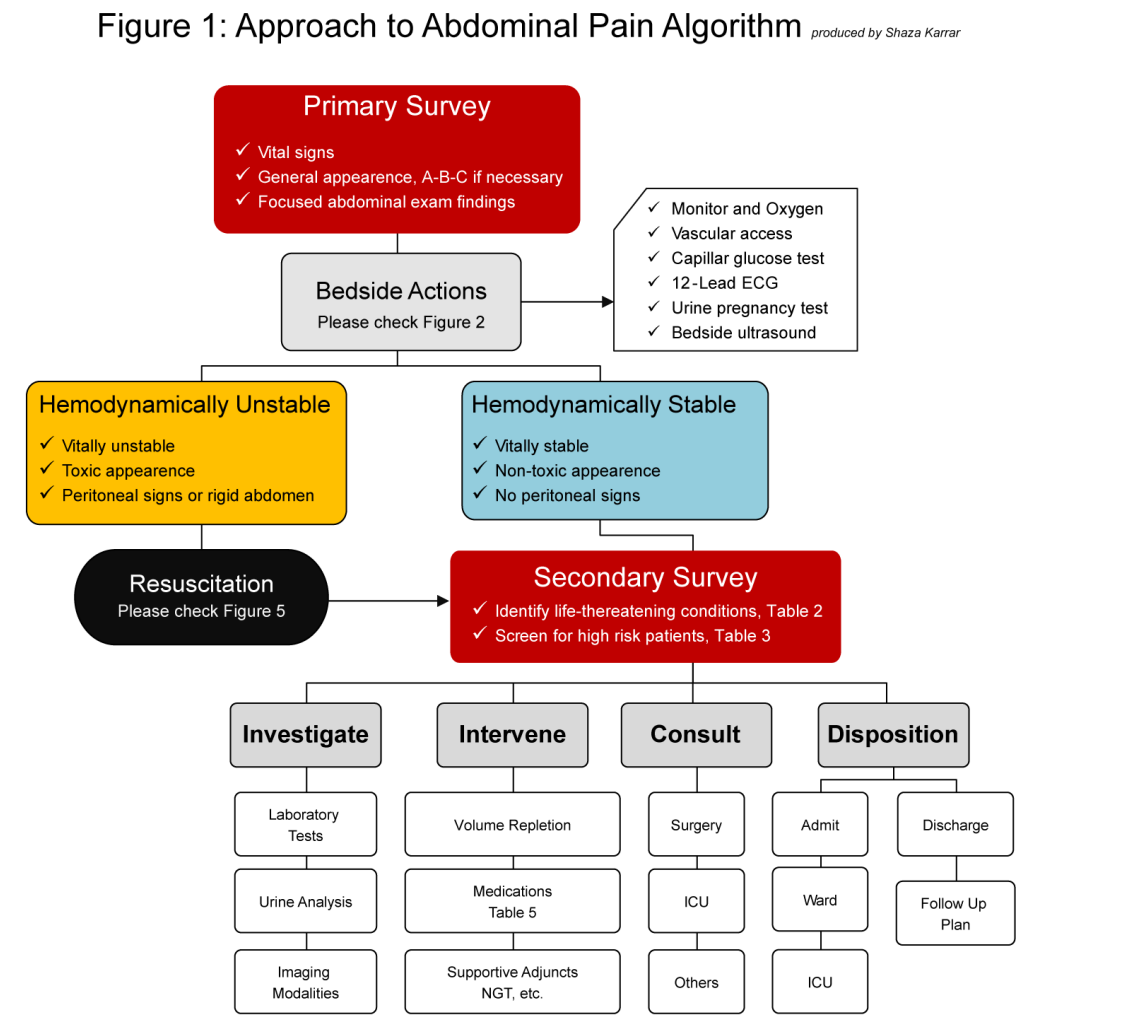
*Produced by Shaza Karrar*

## Critical Bedside Actions

- A systematic approach, starting with a Focused Physical Examination and History Taking is key to reaching a diagnosis and consequently definitive management.
- Assessing the hemodynamic stability of the patient, pain score, and overall distress is a primary step in your approach, as it would set your pace as a clinician gathering bedside information and ultimately your workup and interventions (Figure 3.1).
- Keep in mind: You progress with assessing your patient's stability, history taking and examination, all simultaneously, while advancing with any needed interventions that would stabilize your patient and alleviate his/her pain or distress.
- Do No Harm: Awareness of your limitations is extremely important. Acknowledging when your patient needs urgent assessment by your supervising senior, especially in an

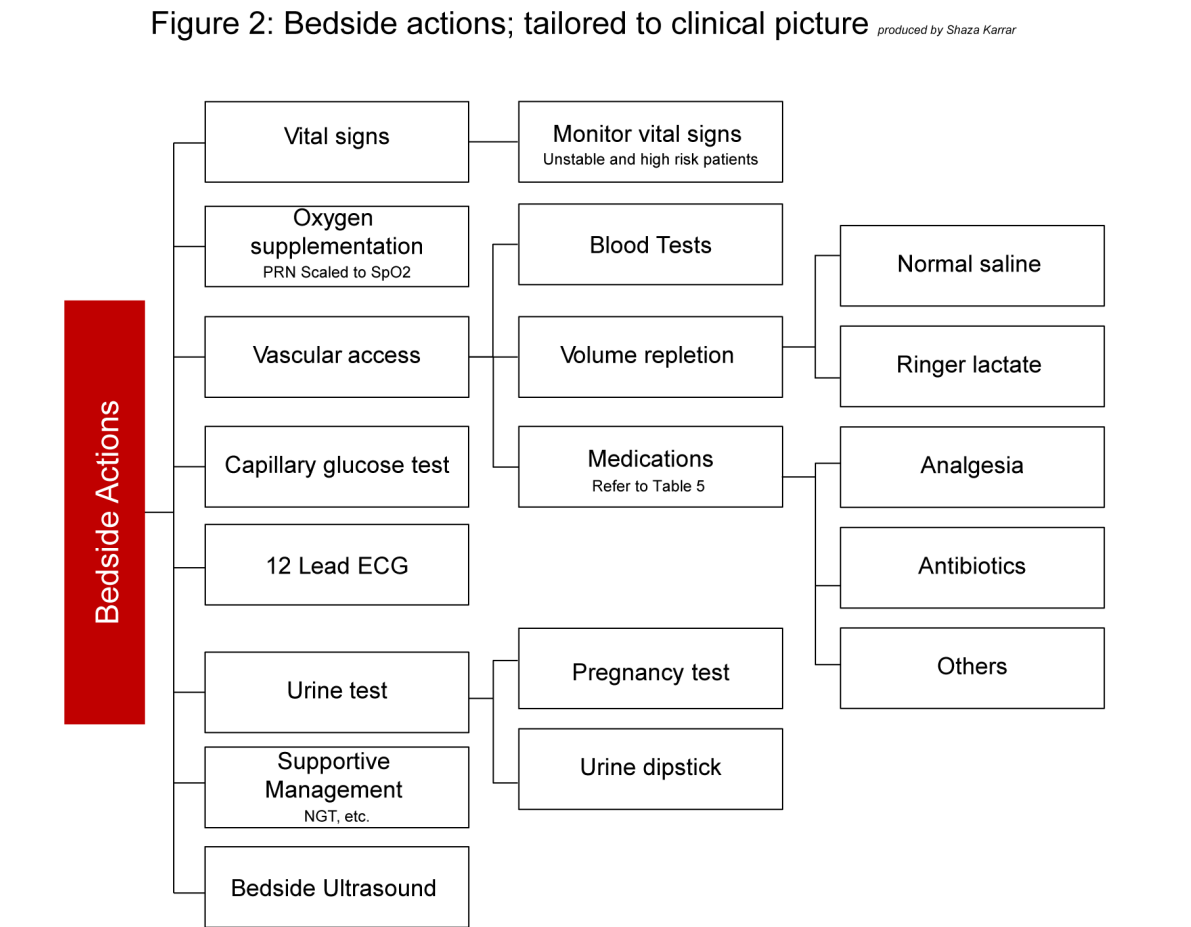
unstable patient, is part of your oath and a cornerstone of your practice during training years.

Figure 3.1 Approach to abdominal pain



👤 Bedside actions are taken in patients presenting with abdominal pain and are tailored to each patient’s clinical picture; those include (Figure 3.2), detailed further in the chapter.

Figure 3.2 Bedside actions




## Differential Diagnoses

Abdominal pain can originate from intra-abdominal and extra-abdominal or non-GI conditions; hence, it’s advisable to be systematic in your approach to narrow down your differential diagnoses. (i.e., Cardiac, GI, infectious, hematologic, urologic, gynecologic, etc.) When diagnosing abdominal pain, the differential diagnoses can be based on anatomic localization of pain (Figure 3.3).

This, in turn, helps direct your approach. An EP should prioritize possible life-threatening conditions in his differential diagnoses, and be mindful of other possible extra-abdominal causes attributable to abdominal pain (Table 3.2).

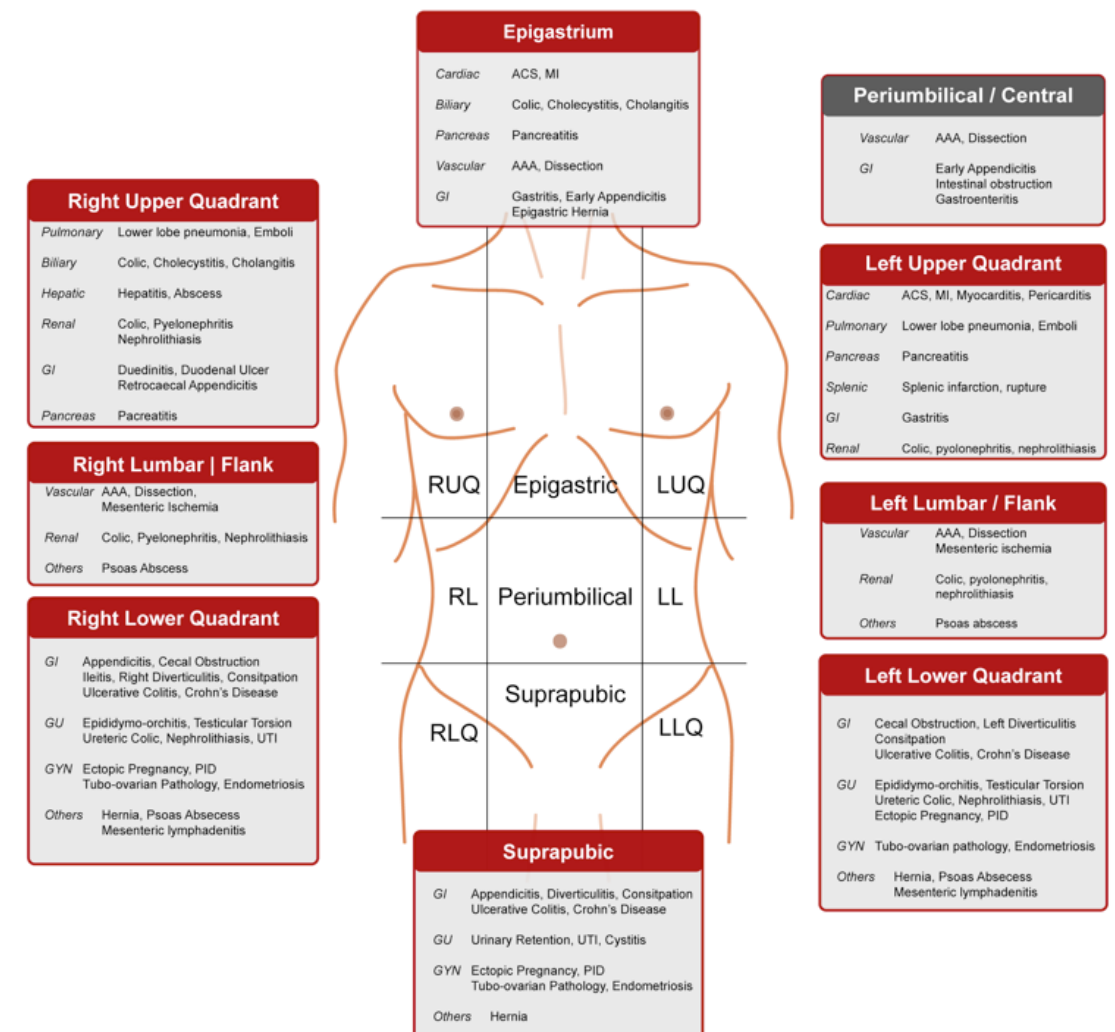
**Table 3.2** Differential diagnoses in abdominal pain

<div>  <b>Table 2: Differential Diagnosis of Abdominal Pain</b> </div>		
Systems	Life/organ-Threatening Conditions	Non-life/organ-threatening Conditions
Cardiac	Myocardial Infarction (MI), Unstable Angina	-
Respiratory	Pulmonary embolism (PE)	Pneumonia
Vascular	Abdominal aortic dissection (AAA), Mesenteric ischemia	-
GI	Appendicitis, Pancreatitis, Perforated viscus, Acute bowel obstruction or ischemia, Volvulus, Diverticulitis, Inflammatory Bowel Disease (IBD); <i>Crohn's Disease or Ulcerative Colitis</i>	GERD, Peptic ulcer disease (PUD), Gastroenteritis, Gastritis, Ileitis, and colitis, Diverticular disease, Constipation/Diarrhea, IBD, Irritable Bowel Syndrome (IBS), Hepatobiliary pathology; <i>Hepatitis, Cholecystitis, etc.</i> , Splenic pathology
Genitourinary	Testicular torsion, Pyelonephritis	Renal colic, Epididymo-orchitis, Urinary Tract Infections Cystitis/Prostatitis
Pelvic	Ectopic pregnancy, Hernias – Strangulated/Incarcerated Placental abruption, Ovarian torsion/ cyst rupture Tubo-ovarian abscess, Threatened abortion	Pelvic inflammatory disease (PID) Cervicitis, Endometriosis
Metabolic	Diabetic-Keto-Acidosis (DKA), Uremia	-
Toxins Medications	Corrosives, Anticholinergics, Narcotics, acetaminophen overdose, Heavy metals	-
Toxins Medications	Corrosives, Anticholinergics, Narcotics, acetaminophen overdose, Heavy metals	-
Inflammatory / Infectious	Rocky mountain fever, Typhoid fever, Psoas abscess	Diarrhea – Infectious, Mesenteric lymphadenitis, Parasitic GI Infections
Tumors Neoplasms	-	Ovarian or colorectal cancer Leukemia
Hematologic	Sickle cell crises	-
Autoimmune	-	Henoch-Schonlein purpura, Systemic Lupus Erythematosus (SLE), Familial-Mediterranean fever
Others	Glaucoma, Trauma	Abdominal wall spasm

Produced by Shaza Karrar

**Figure 3.3** Differential diagnoses according to location

Figure 3: Differential Diagnoses According to Localization of Abdominal Pain produced by Shaza Karrar



Keep in mind that the following list of differentials is not exhaustive and should be correlated with the localization of abdominal pain (Figure 3.3).



# History Taking and Physical Examination Hints

## History Taking Hints

A properly focused history is of paramount importance, as it guides your physical examination, differential diagnoses, critical interventions, work up, and ultimately your disposition. Specific history findings can lead to prompt and accurate diagnosis and management.

### History taking should cover the following

- 📌 General impression: level of distress, stability and pain scale.
- 📌 Patient Demographics: Gender, age.
- 📌 Onset and progression of pain: Sudden, gradual, episodic/intermittent, continuous.
- 📌 Localization of pain and radiations (Figure 3.3).
- 📌 The character of pain: Dull, sharp, colicky, stabbing, burning
- 📌 Severity: Pain Scale – mild, moderate, severe, or a scale from 1 to 10
- 📌 Alleviating and aggravating factors: Relieved by eating, sitting up, or worsens after eating, lying supine, movement, and coughing, etc.

- 📌 Associated symptoms: Be systematic – Fever, nausea, vomiting, loose-motions, melena, jaundice, dysuria, vaginal discharge/bleeding, shortness of breath, etc.
- 📌 Past medical, surgical and gynecologic/obstetric history: Known GI pathologies, chronic comorbid, recent trauma, previous similar complaint, previous surgeries, LMP
- 📌 Social history and Travel history: Alcohol consumption, illicit drug abuse, occupational hazards or exposures
- 📌 Medications and allergies history: Anticoagulants, corticosteroids, contraceptives.
- 📌 Others: Clues of an atypical presentation – considering Extra-Abdominal and non-GI pathologies including weight loss, unspecific systemic symptoms, etc.

Always try to screen for high-risk patients; certain history findings can help narrow your differential diagnoses. Table 3 demonstrates history findings, coinciding with their potential differentials.

## Physical Examination Hints

A focused and systematic Physical Examination (P.E.) aims at verifying your clinical impression constructed from your history findings; it also aids in exposing unforeseen findings that may make you reassess your differential diagnoses and approach. Hence, an EP must have in mind specific working differentials obtained from the history before examining the patient (Figure 3.3

and Table 3.3). Make sure that your patient is comfortable with adequate pain relief as necessary, suitably positioned and appropriately exposed from nipples to lower abdomen/pubic-symphysis in a properly private environment. In the case of children, the presence of family members can aid your examination. Assessment of vital signs, hemodynamic stability, and signs of shock should be noted, keeping in mind that normal vital signs would not rule out a life-threatening condition.

**Table 3.3** History findings

iEM Table 3: History Findings and Potential Diagnoses	
History Findings	Possible Differential Diagnosis
Females of Childbearing age, Associated vaginal bleeding	Ectopic pregnancy, miscarriage, abortion
Elderlies, age >65, History of heart or peripheral vascular diseases, or atrial fibrillation (AF)	AAA, mesenteric Ischemia – <i>High-risk Patients</i>
Pain out of proportion to clinical picture in elderly	Mesenteric ischemia
Immunodeficiency, i.e. HIV, Chronic Steroid, etc.	Occult infections – <i>High-risk Patients</i>
Abdominal surgical history	Adhesion or obstruction
Sudden onset of severe pain	MI, Perforated viscus, Ruptured AAA/ectopic pregnancy/ovarian cyst – <i>High-risk Patient</i>
Character of colicky pain	Gastroenteritis, obstruction, biliary/ureteral colic
Alleviated positional pain by sitting forward, alcohol abuse, or radiation of pain from epigastrium to back	Pancreatitis
Radiation from loin to groin	Passing kidney stones
Patient lying flat and still, with discomfort and aggravation of pain with minimal movement	Peritoneal irritation
Adolescent males	Testicular Torsion
Alcohol abuse	Pancreatitis, Hepatic cirrhosis
Considered by all age groups	Appendicitis

*Produced by Shaza Karrar*

Henceforth, a P.E. would be based on a focused and systematic approach, consisting of:

General appearance and vital signs assessment:

- Note any instability in vitals, consciousness level, the posture of the patient, hydration status and signs of pallor, jaundice.

Examination of extra-abdominal systems:

- Entails an assessment of the cardiorespiratory functions.
- Other extra abdominal systems of suspicion attributing to the patient history and clinical picture.

Focused abdominal examination:

- Inspection: Distention, surgical scars, masses, distended veins, skin discoloration.
- Palpation:
  - The light then deep palpation begins with the opposite non-tender quadrant, progressing through all quadrants.
  - Assess for tenderness, guarding, rebound tenderness, hepatosplenomegaly, and masses; for instance, a rigid, tense abdomen with involuntary guarding is highly indicative of peritonitis. Look for costovertebral angle tenderness and palpate
  - Look for costovertebral angle tenderness and palpate bimanually for renal masses.
- Percussion: Dullness for ascites or Resonance.

- Auscultation: Hyper/hypoactive or absent bowel sounds and vascular bruit.
- Specific signs and maneuvers: (Table 3.4)
  - For an overview of the focused Abdominal Examination, please watch the following video.
  - Serial abdominal examinations are important to reassess your patient's progress and response to treatment.
  - Rectal examination: In suspected GI bleeds, perianal and prostatic disease, foreign bodies, and impacted stools.

- Examination of hernial orifices: All should be cleared; easily missed in the exam.
- Genital and pelvic examination:
  - Pelvic exam in all females with lower abdominal pain and query pelvic pathologies.
  - Genital exam in males with possible testicular pathologies and hernias.

# Emergency Diagnostic Tests and Interpretation

## Bedside tests

- 12-lead ECG
  - Initial screening for cardiac pathologies (i.e., Acute Coronary syndrome; MI, AFib, Digoxin toxicity, etc.), especially in patients presenting with epigastric pain, cardiovascular risk, and elderlies with poorly localizable pain. Consider follow-up cardiac enzymes in high-risk patients and abnormal ECG findings.

What is your opinion about below ECG in a patient with abdominal pain?

**Table 3.4** Abdominal signs

iEM Table 4: Specific Abdominal Signs and Maneuvers		
Sign	Definition	Differential Diagnosis
Murphy's sign	Suspension in deep inspiration upon palpation of the RUQ due to tenderness	Cholecystitis
Kehr's sign	Left shoulder tip pain, specifically when the patient lies supine, due to irritation of the peritoneum	Ruptured Spleen or injury Ruptured ectopic pregnancy
McBurney's sign	Tenderness upon deep palpation of McBurney's Point; found midway between the umbilicus and the right anterior iliac spine	Appendicitis
Psoas sign	Irritation of the iliopsoas group; at extension of the right hip while patient lies on left side elicits abdominal pain	
Obturator's sign	Internally rotating a flexed right hip elicits abdominal pain	
Rovsing's sign	RLQ pain upon palpation of LLQ	
Heel-drop sign	Dropping heels on the ground after standing on tiptoes, or forcefully striking the patient's heel elicits RLQ pain	Peritoneal irritation
Cough sign	Tenderness when patient is asked to cough	
Pulsatile masses	Pulsating mass upon palpation of the abdomen	
Cullen's sign	Periumbilical ecchymosis	
Grey-Turner's sign	Flanks ecchymosis	Retroperitoneal hemorrhage; Hemorrhagic pancreatitis, Ruptured AAA

Please check Chai Chan and Seow (2015) and White and Counselman (2005) in references to learn more about specific findings and maneuvers in abdominal pain.  
Shaza Karrar

**Image 3.1 ECG**



- Urine Pregnancy test: All females of childbearing age, regardless of history findings
- Urine analysis: Signs of hematuria or Urinary Tract Infections (UTI).
- Capillary Glucose test: Hyper/Hypoglycaemia and DKA
- Ultrasound
  - Considered the new EP stethoscope – bedside, non-invasive, yet operator-dependent
  - Aids at assessing solid abdominal organs, the presence of intraperitoneal fluid, vascular and hemodynamics of the patient

### Laboratory tests

- Complete-Blood-Count: Leukocytosis, Hemoglobin level, Platelets count
- Electrolytes: Correct any derangements caused by fluid losses.

- Blood-Urea-Nitrogen(BUN): Assess dehydration.
- Creatinine / Renal Function: Assess renal functions, dehydration, and record a baseline in case of needed contrast imaging studies to prevent possible contrast-induced-nephropathy.
- Liver function test (LFT): Liver and gallbladder pathologies
- Amylase: Increased in most intra-abdominal pathologies.
- Lipase: Levels twice the normal is highly indicative of pancreatitis, joined with elevated LFT, could raise the suspicion of Gallstones pancreatitis.
- Inflammatory markers: CRP or Procalcitonin
- Coagulation profile: Patient on anticoagulation (i.e., Warfarin for AF) – if reveals sub-therapeutic levels, would raise suspicion of mesenteric ischemia, or if supra-therapeutic, would raise suspicion of active bleed, as well a standard test for possible surgical interventions; in need of reversal.
- Quantitative hCG Blood: Confirmatory after a urine pregnancy test
- TestType and screen: For all patients possibly proceeding for surgical interventions
- Rh Status: All female patients with possible ectopic pregnancy



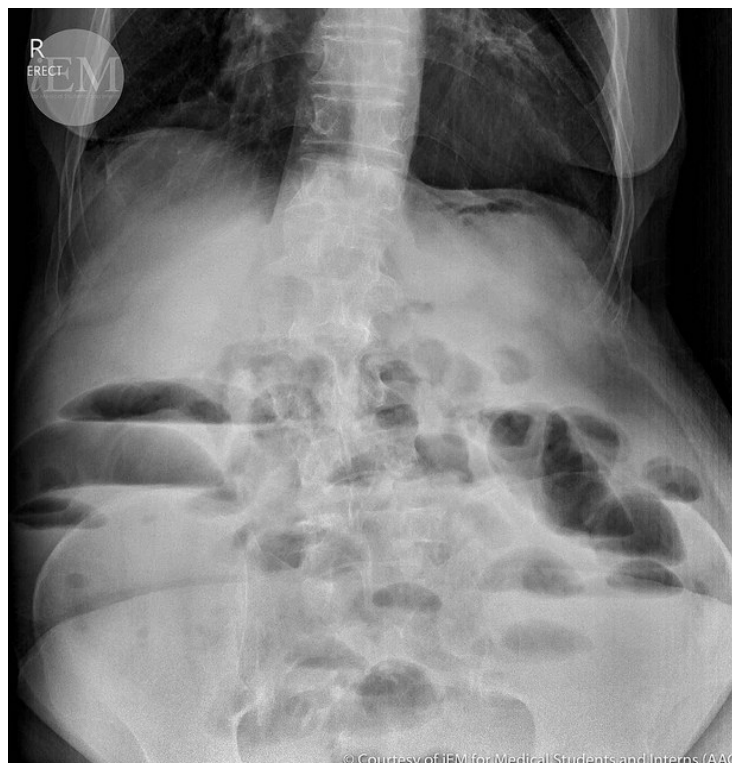
## Imaging modalities

### X-rays

- An initial imaging modality, quick yet not very sensitive
- Chest X-rays: Delineate Air under the diaphragm in perforated viscus or pneumonia.
- Abdominal X-rays: Usually in erect and decubitus positioning; looking for Bowel distention, air-fluid levels, obstruction, foreign bodies.

What is your opinion about below abdominal x-ray in a patient with abdominal pain?

**Image 3.2** Abdominal x-ray

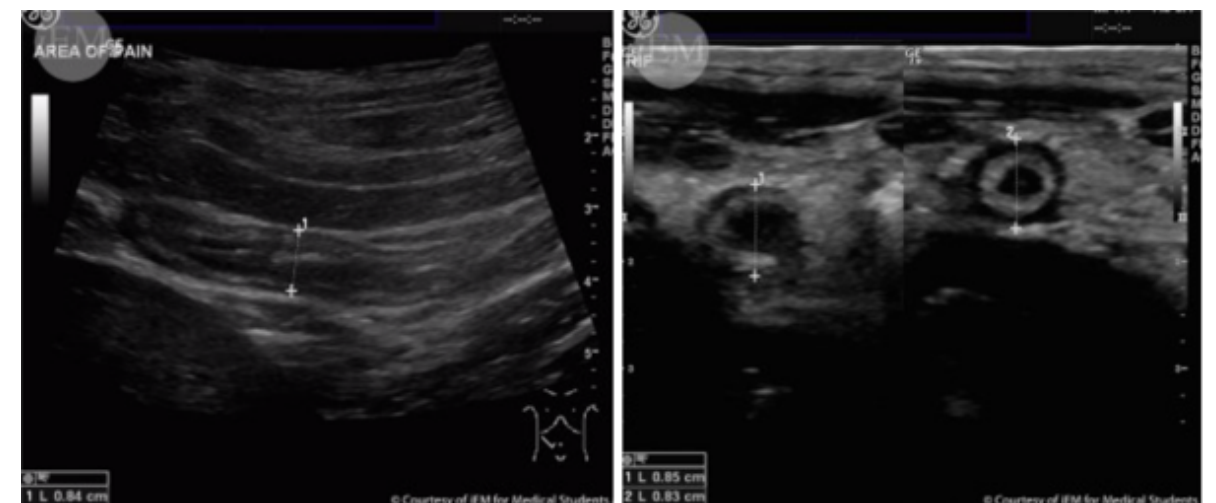


### Ultrasound (US)

- Extended sonographic studies can further your assessment; considered the study of choice in pregnant women.
- Abdominal US: Evaluation of biliary tract pathologies, intraabdominal organs, and free intraperitoneal fluid, Intussusception, appendicitis, etc.

What is your opinion about below ultrasound samples in a patient with abdominal pain?

**Image 3.3** Abdominal US, RLQ



- Kidney-Ureter-Bladder KUB US: Nephrolithiasis, hydronephrosis, urine retention, etc.
- Pelvic or Obstetric US: Tubo-ovarian pathologies, intrauterine and ectopic pregnancy, fibroids, etc.
- Testicular US: Testicular torsion, epididymo-orchitis



- Vascular US Studies: assessment of the abdominal aorta

### Computed tomography (CT) scan: (With/Without Contrast, Angiography)

- Highest in sensitivity and specificity in detecting most common pathologies
- Ionizing and considered limited in the case of contrast allergies, pregnancy, or pre-existing renal insufficiency predisposing the patient to contrast-induced nephropathy.

### Magnetic resonance imaging (MRI):

- Limited use in the ED, considered in pregnant patients of inconclusive US findings.

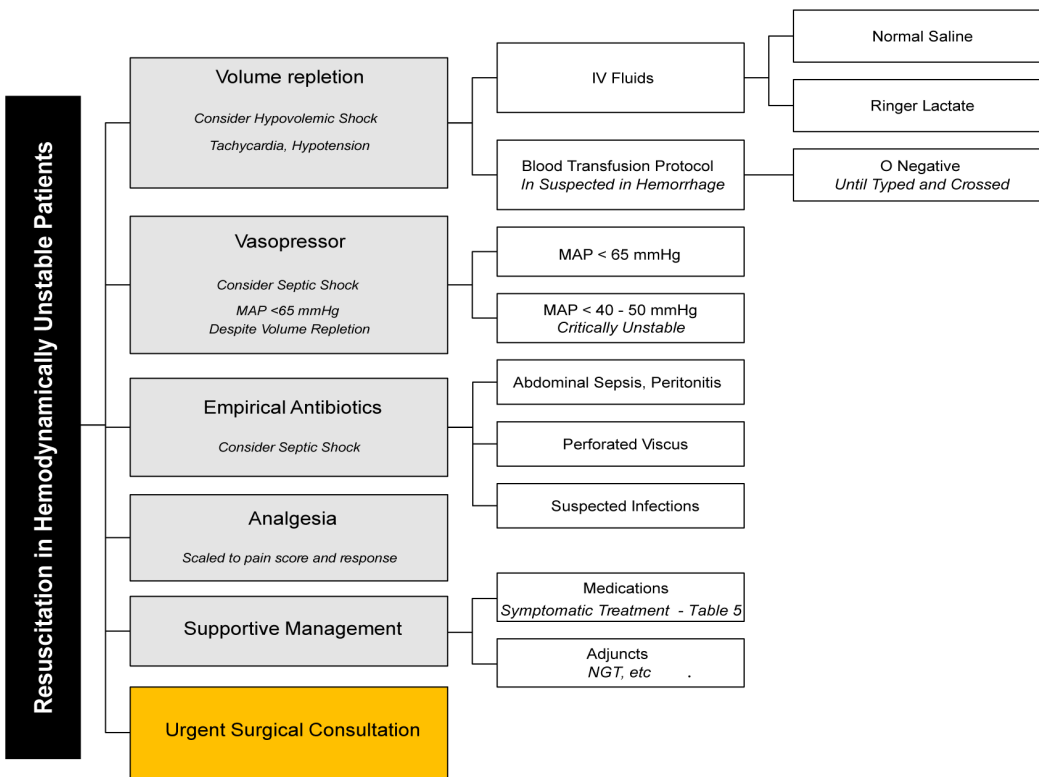
## Emergency Treatment Options

### Initial Stabilization

- 📍 Primary survey: Hemodynamic stability assessment subsequently sets the pace of your approach (Figure 3.1).
- 📍 Secondary survey: Identify life-threatening conditions (Table 3.2), and screen for the high-risk patients, and special age groups (Table 3.3).
- 📍 Hemodynamically unstable patients should be resuscitated without any delay, entailing the following keystones (Figure 3.4).

Figure 3.4 Resuscitation of unstable patient

Figure 4: Resuscitation of hemodynamically unstable patients produced by Shaza Karrar



- 📍 Appropriate surgical consultations should be sought in a timely manner, especially in the hemodynamically unstable patient and surgical conditions in need of an intervention.
- 📍 A hemodynamically stable patient should be properly worked up and reassessed frequently, as he/she may deteriorate and become unstable.
- 📍 Patients with possible peptic ulcer disease (PUD) and gastritis can benefit from a “GI cocktail,” typically

constituted of a combination of antacid, viscous lidocaine, and antispasmodics.

- Otherwise, stable patients who are responsive to treatment, with conditions of low acuity, can be fit for discharge from the ED with an appropriate disposition and follow-up plan.

## Medications

- Analgesics should be tailored to each patient's clinical picture, pain score, and response, with an aim to relieve his distress and pain to a manageable level, making him more comfortable and cooperative for the abdominal exam and reassessment (Table 3.5).

**Table 3.5** Table 5. Medications in abdominal pain

iEM

Table 5: Medications in Abdominal Pain

Indication	Medication	Dosage Descriptions		Pregnancy Category
		Adult   >13 years OR >50 Kg	Pediatric   2 -12 years OR <50 Kg	
Antipyretics: Control of Fever, Analgesic as well.				
Fever and mild to moderate pain.	Paracetamol	1000 mg IV/PO q4-6hr Max 1000mg/dose 4000 mg/day	15 mg/kg IV/PO q4-6hr Max 4mg/kg/day	Class C
Analgesia: Tailored to Clinical Picture, Pain Score, and Scaled to Response.				
Mild Pain	Paracetamol	As stated above	As stated above	Class C
	Ketorolac NSAIDs	<ul style="list-style-type: none"> <li>IM: 60 mg Once a day or 30 mg every 6 hours</li> <li>IV: 30 mg as a single dose or 30 mg every 6 hours</li> <li>Maximum: 120 mg/day</li> </ul>	<ul style="list-style-type: none"> <li>IM: 30 mg once a day or 15 mg every 6 hours</li> <li>IV: 15 mg as a single dose or 15 mg every 6 hours</li> <li>Maximum: 60 mg/day</li> </ul>	Class C
	<ul style="list-style-type: none"> <li>NSAIDs - Ketorolac: As stated above.</li> <li>Paracetamol with an adjunctive opioid analgesic.</li> </ul>			
Mild to Moderate Pain	Morphine Opioid	<ul style="list-style-type: none"> <li>0.05 - 0.10 mg/kg IV</li> <li>Typical adult dose 2 to 5 mg IV; given every 5-15 minutes to response.</li> </ul>	<ul style="list-style-type: none"> <li>0.05 to 0.3 mg/kg every 3 to 4 hours as needed</li> <li>Max 10 mg per dose</li> </ul>	Class C
Moderate to severe pain	Fentanyl Opioid	<ul style="list-style-type: none"> <li>to 0.3 mcg/kg IV</li> <li>Typical adult dose 10 to 25 mcg, given in five-minute intervals to response</li> </ul>	<ul style="list-style-type: none"> <li>0.5 to 1mcg/kg/dose given in five-minute intervals to response</li> </ul>	Class C
Antiemetic: Symptomatic treatment of nausea and vomiting, i.e. Metoclopramide, Ondansetron				
Antibiotic: Tailored according to provisional diagnosis.				

Please check UpToDate.com and Drug ratings in pregnancy (US Food and Drug Administration) in references to learn more about medications in abdominal pain

Shaza Karam

Please check UpToDate.com and Drug ratings in pregnancy (US Food and Drug Administration) in references to learn more about medications in abdominal pain. Shaza Karrar

- Abdominal pain should be addressed with liberal analgesia, including the use of opioids, as it has been recently proven that they do not alter physical exam findings or increase the number of incorrect management decisions.
- Opiate-dependent patients and chronic users may need higher doses for an adequate response.
- Always monitor patients for respiratory depression with opioids and always consider dose adjustments in geriatrics and patients with renal and hepatic impairments.
- NSAIDs like Ketorolac are suitable for biliary and renal colic but not in PUD.

## Procedures: None

## Pediatric, Geriatric, Pregnant Patient and Other Considerations

### Pediatric Patient

- The list of differential diagnoses tends to rearrange in acuity according to the age at presentation in pediatric patients. For example:
- 0 to 3 months: testicular torsion, necrotizing enterocolitis, incarcerated hernia and pyloric stenosis
- 3 months to 3 years: intussusception, vaso-occlusive crisis, UTI and toxic megacolon, among those just stated above

- 3 years to adolescence: appendicitis, mesenteric lymphadenitis, DKA and toxic ingestion, as well among those just stated above
- When it comes to examining for peritoneal sign in pediatrics, children can be asked to jump up and down as an indirect means of inciting peritoneal irritation.

## Geriatric Patient

- Elderly and immunocompromised patients deserve special attention as a higher risk group compared to their younger counterparts; they are more likely to present late in the disease process and with atypical presentations.
- The elderly commonly present with multiple comorbidities, difficult communication skills, and vague symptoms that are not reflective of the actual severity of their disease.
- It's important to establish their medication and allergies history (Anticoagulants, etc.)
- They cannot tolerate volume loss and hence can deteriorate easily, and their abdominal exam is often unspecific; however, they're the most likely population to have surgical emergencies.

## Pregnant Patient

- Females of childbearing age presenting with abdominal pain are presumed to have an ectopic pregnancy until proven otherwise, regardless of their history.
- Pregnant women are at risk of the same abdominal conditions as all other patients, but they propose special considerations.
- Restricted imaging necessitates astute work up and reassessment.
- Consider first-trimester abortion in abdominal pain in a pregnancy < 20 weeks of gestation.
- In pregnant women well advanced in their pregnancy, consider obstetric emergencies, such as Preterm-Labor, Placenta Previa and abruption, Uterine Rupture and HELLP syndrome.

## Other considerations

- Appendicitis should be considered in all patients presenting with acute abdominal pain, refer to Alvarado Score.
- Abdominal trauma, blunt and penetrating, results in a myriad of intraabdominal injuries that shouldn't be taken lightly but well investigated and addressed.

## Disposition Decisions

### Admission criteria

- Hemodynamically unstable patients require admission to the Intensive Care Unit (ICU), especially post possible surgical interventions.
- Elderly patients with multiple comorbidities or anticipated clinical course deterioration require an ICU admission as well or a High Dependency Unit (HDU) if available.
- Hemodynamically stable patients found to have surgical conditions can be admitted to a general ward.
- The conservative and low threshold for admission is strongly advised and advocated in the high-risk groups.
- Certain stable patients might need to be admitted for further evaluation and planned interventions.

### Discharge criteria

- Patients who are responding to treatment, with a resolution of symptoms, without signs of underlying life-threatening conditions, can be discharged.
- Always instruct your patients to return to the ED in the case of progression of abdominal pain or worsening general condition, which is not getting relieved, such as persistent vomiting, jaundice, fever, etc.

- Patients with an unclear etiology of pain who are stable enough for discharge should be reassessed again within 12 to 24 hours; hence, a follow-up plan should be instituted and emphasized to the patient.

### Referral

- Follow up plans in patients that are discharged decrease high morbidity and mortality, a chance of a missed diagnosis, and decrease unnecessary ED presentations of benign and low acuity abdominal pain.
- Follow up plans are essential and have to be arranged for certain patients. Those include:
  - Patients in need of the re-evaluation of their symptoms' progression and resolution; particularly patients at higher risk.
  - Patients with an unclear pathology that needs a further workup by multidisciplinary services – vascular, urology, OB/GYN, gastroenterology, etc.
  - Patients with known GI pathologies or comorbidities, who did not follow-up, are in need of reinstitution of their primary or specialty care.
  - Pregnant women should resume their primary care obstetricians, with an advised reassessment as soon as possible post discharge.

- Yet pregnant women are strongly instructed to return to the ED in case of recurred, progressing or persistent symptoms, especially in cases of uncorrected volume loss due to vomiting and loose motions, vaginal bleeding/discharge, and abdominal pain.

**References and Further Reading**, click [here](#).



# Altered Mental Status

---

by Murat Cetin, Begum Oktem, Mustafa Emin Canakci

## Case Presentation

*An 80-year-old female presents to the emergency department with a tendency to sleep (altered mental status), failure in recognizing people and answering questions. She is a nursing-home inhabitant. The caregivers express she was feverish and fatigued for several days now, but her mental problems have recently begun. The patient has a history of hypertension and diabetes mellitus. Her only routine medications are angiotensin-converting enzyme inhibitors (ACE inhibitors) and insulins. Vital Signs: Blood Pressure: 110/70 mmHg, Heart rate: 110 bpm, respiratory rate: 20 rpm, temperature: 38.8 degrees Celsius, peripheral capillary oxygen saturation: 98%, finger-stick blood sugar: 95 mg/dL. Physical Exam: She is in mild distress, lethargic and confusional with no lateralizing signs. The pupils are reactive to light and equal in size. On a Glasgow Coma Scale, she is registered at 12 (E3,*



Audio is available [here](#)

*M5, V4) and she had neck stiffness. The heart is irregularly tachycardic with no abnormal cardiac sounds. The breath sounds are clear and equal bilaterally. The abdomen is soft, non-tender, non-distended. Skin: warm, dry, no rash. A lumbar puncture is performed to diagnose or exclude meningitis.*

## General Approach and Critical Bedside Actions

### General Approach and Key Concepts

The state of consciousness is a sum of arousal and cognition. Arousal refers to awareness of the self and the environment. The Ascending Reticular Activating System in the brainstem modulates arousal. The Cognition is the combination of orientation, reasoning, and memory. The cerebral cortex houses the cognition centers. In sum, the normal state of consciousness requires a properly functioning brain stem and cerebral cortex.

Altered mental status may result from any changes in;

- Arousal,
- Cognition,


- A combination of these two functions.


The altered mental state may mean coma, confusion, aggression, personality alteration, or difficulty in awakening. Approximately 3% of patients in the emergency department have impaired mental status. In the elderly patients, this rate is between 10% and 25%. 85% of patients have metabolic and systemic diseases.

Dementia and delirium should be differentiated in patients with altered mental status. Metabolic, infectious and vascular pathologies should be evaluated.

### Critical Bedside Actions

The initial evaluation must start with the evaluation of airway, breathing, and circulation (ABC) sequence. Life-threatening situations require rapid intervention. During the initial assessment, the reversible causes must be addressed and managed such as hypoglycemia or opioid overdose. A continuous cardiac monitoring and vascular access must be established as soon as possible.

 A (Airway): Hypoxia is a reversible cause of altered mental status. Ensure the airway is open and protected. If not, secure the airway and give oxygen if necessary. We prefer oxygen saturation maintained above 94%.

 B (Breathing): Inadequate ventilation may cause hypercarbia. If the respirations are inadequate or superficial, perform bag-

mask ventilation (BVM) or endotracheal intubation. The combination of altered mental status and respiratory depression may suggest narcotic/opioid overdose.

🔊 C (Circulation): Hypoperfusion may cause altered mental status due to decreased oxygen and glucose in the brain. Check the distal pulses, blood pressure, and cardiac rhythm. Capillary refill time, skin color and temperature may also help to understand possible shock situation. If the patient is hypotensive, administer IV fluids and investigate the cause.

🔊 D (Mini neurological evaluation): Assess Glasgow Coma Score (GCS) or AVPU quickly. Check if the pupils are equal and reactive to light. Administer benzodiazepines (lorazepam or diazepam, based on availability) in case of ongoing seizure activity. Cervical stabilization should be provided if trauma is suspected.

🔊 E (Exposure): The findings should be evaluated in terms of trauma, transdermal drug tapes, dialysis intervention area, sources of infection and petechiae.

🔊 Glucose level, ECG should be performed. Bedside ultrasound (**eFAST** or **RUSH** protocols) should be added to the investigation of patients with shock or trauma.

🔊 The level of hemodynamic stability of the patient sets the pace of the investigation. A systematic approach, starting with

history taking and focused physical examination is key to reaching a diagnosis and consequently definitive management.

## Differential Diagnoses

Metabolic, infectious, toxicological, endocrine, hypoxic conditions may cause altered mental status. The mnemonic “AEIOU-TIPS” may help to remember most common causes of altered mental status (Table 3.6).

## History Taking and Physical Examination Hints

### History Taking

Obtain patient’s medical history directly from the patient. Determine if the level of awareness is decreased. If the patient is unable to provide the necessary information:

- Ask a family member, caregiver, or medical personnel
- Check for medical alert identification
- Ask for medical information sheet (i.e., on the refrigerator)
- Ask surrounding environment (i.e., living quarters, alcohol bottles or drug paraphernalia).

**Table 3.6** Mnemonic AEIOU-TIPS for Altered Mental Status

MNEMONIC	THINGS TO CONSIDER
Alcohol	Alcohol levels, serum osmoles
Epilepsy/ Endocrine/ Electrolytes/ Encephalopathy	EEG, referral to neurology, TFTs, cortisol, chemistry panel, LFTs/NH3
Insulin	Glucose
Oxygen/ Opiates	SatO2%, ABG, hypoxia makes agitation, hypercarbia makes somnolence Look for needle marks
Uremia	BUN/Cr Things changing serum osmolarity affect mental status. Uremia, Sugar, Alcohol are common ones
Trauma/ Temperature	CT Head, C-Collar, CT C-Spine
Infection	CBC, BCx, UA, UCx, CXR, LP/CSF Sepsis and CNS infections are more important. But, even simple fever may cause AMS in elderly and kids
Poisoning/ Psychosis	Drug Levels (e.g. lithium, digoxin)
Shock/ Stroke/ SAH/ Space occupying lesion	ECG, Troponin, CT Head, LP

If the normal state of the patient is unclear, all changes must be evaluated as if they are acute. Strokes, seizures, cardiac events, intoxication, psychiatric disorders cause sudden changes, whereas; infections, metabolic disturbances, or an expanding intracranial mass may cause gradual changes.

Altered mental status is a result, not a diagnosis. The diagnosis is based on clinical suspicion. The cause may be transient (seizure) or permanent (stroke), benign or life-threatening. If not treated timely and accurately, most causes may be mortal or cause neurologic sequelae. The systematic and structured approach makes diagnosing and management easier.

## Physical Examination Hints

A focused and systematic physical examination aims at confirming the clinical impression formed by the history. It also aids exposing unexpected findings that may make the clinician reassess the differential diagnoses and approach. Repetitive examinations should be performed to track changes.

- Vital signs should be evaluated very carefully in terms of hypotension, hidden shock, hypoxia, respiratory rate and pattern, and temperature.
- Head: Signs of trauma, pupils' size and reaction to light, cterus, pale conjunctiva
- The fundoscopic exam may show hemorrhage, papilledema
- Neck: Rigidity, bruits, thyroid enlargement
- Heart and Lungs may show heart failure, pneumonia findings
- Abdomen: Organomegaly, ascites
- Extremities: peripheral cyanosis

- Skin: Diaphoretic/dry, rash, petechiae, ecchymoses, splinter hemorrhages, needle tracks
- Neurologic exam should be done in order of GCS (Table 3.7), FOUR score (Table 3.8), pupil dimensions, neck stiffness, lateralizations . In the secondary evaluation, the full neurological exam should be applied. The mental status exam should be the main part of neurological exam and repeated as needed.

**Table 3.7** Glasgow Coma Scale

EYE OPENING	BEST VERBAL RESPONSE	BEST MOTOR RESPONSE
4: Spontaneously	5: Oriented and converses	6: Obeys command
3: To verbal command	4: Disoriented and converses	5: Localizes pain
2: To pain	3: Inappropriate words; cries	4: Flexion withdrawal
1: No response	2: Incomprehensible sounds	3: Flexion abnormal (decorticate)
	1: No response	2: Extension (decerebrate)
		1: No response

Glasgow Coma Score (GCS) (Modified from Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. *The Lancet*, 304(7872), 81-84.) - Please read this article to get more insight regarding GCS.

**Table 3.8** Full Outline of Un-Responsiveness (FOUR) Score

EYE RESPONSE	MOTOR RESPONSE	BRAINSTEM REFLEXES	RESPIRATION
4: Eyelids open or opened, tracking, or blinking to command	4: Thumbs-up, fist, or peace sign	4: Pupil and corneal reflexes present	4: Not intubated, regular breathing pattern
3: Eyelids open but not tracking	3: Localizing to pain	3: One pupil wide and fixed	3: Not intubated, Cheyne-Stokes breathing pattern
2: Eyelids closed but open to the loud voice	2: Flexion response to pain	2: Pupil or corneal reflexes absent	2: Not intubated, irregular breathing
1: Eyelids closed but open to pain	1: Extension response to pain	1: Pupil and corneal reflexes absent	1: Breaths above ventilator rate
0: Eyelids remain closed with pain	0: No response to pain or generalized myoclonus status	0: Absent pupil, corneal, and cough reflex	0: Breaths at ventilator rate or apnea

Gujjar AR, Jacob PC, Nandhagopal R, Ganguly SS, Obaidy A, Al-Asmi AR. Full Outline of UnResponsiveness score and Glasgow Coma Scale in medical patients with altered sensorium: interrater reliability and relation to outcome. *J Crit Care*. 2013 Jun;28(3):316.e1-8. doi: 10.1016/j.jcrc.2012.06.009. Epub 2012 Aug 9. PubMed PMID: 22884530. - Please read this article to get more insight about this score.



## Emergency Diagnostic Tests and Interpretation

Differential diagnosis of altered mental status is broad, and diagnostic tests should be targeted to suspected underlying pathologies. Rather than a single specific algorithm, a ruling in and out approach should be followed.

### Bedside tests

- Rapid glucose: Quick and easy. Hypoglycemia is an emergent cause and can be ruled out with this simple test, just in seconds. Glucose level should be measured at the bedside to understand hypo/hyperglycemia problems including diabetic ketoacidosis and hyperosmolar hyperglycemic state.
- ECG may help to understand arrhythmias and some toxic effects of drugs such as TCA overdose.
- Bedside US such as **eFAST** for trauma patients and **RUSH** protocol for nontraumatic hypotensive patients can be valuable to understand the underlying causes. Some other US applications can be used for altered mental status cases such as optic nerve sheath diameter measurement to understand increased intracranial pressure.

### Laboratory tests

Many laboratory tests can help the management of altered mental status cases. These tests and their possible findings were given below.

**Arterial/venous blood gas investigation** is necessary for many critically ill patients to understand acidosis/alkalosis, hypercarbia, hypoxemia, carboxy-hemoglobinemia, methemoglobinemia, lactate, and base excess situation.

**Complete blood count** can be used to search for Anemia/ Polycythemia in hyperviscosity related pathologies, Leukopenia/ Leukocytosis in Infection and sepsis, Thrombocytopenia / Thrombocytosis in spontaneous intracranial hemorrhage.

Renal function tests (RFT) for uremia suspicion caused by acute or chronic renal failure. RFT can also be necessary to understand patients' baseline before to order contrast-enhanced imaging or use some drugs.

Liver function tests are useful in the suspicion of hepatic failure, hepatic encephalopathy or biliary tract problems.

Electrolytes are essential to evaluate altered mental status. There are many electrolyte abnormalities can change the level of consciousness such as hypo/hyponatremia, hypo/hyperkalemia, hypo/hypercalcemia.

Urinalysis shows ketone bodies of diabetic ketoacidosis and provides information about urinary tract infection.

Thyrotoxicosis and myxoedema coma are important endocrine-related causes of altered mental status. Therefore thyroid function tests can be valuable in some circumstances.

Cardiac enzymes can be necessary because myocardial infarction may cause a low ejection fraction or trigger arrhythmias and patients may show altered mental status.

Also, when needed:

- Drug screening for a suspected overdose
- Levels of specific medications such as antiepileptics, antipsychotics, digoxin, warfarin
- Ethanol level, levels of toxic alcohols
- Cerebrospinal fluid (CSF) tests after a lumbar puncture (LP)
- Cultures (Blood, urine, CSF, etc.)

### Imaging modalities

- **Head CT:** Non-contrast CT for ruling out hemorrhage, mass effect, edema.

What is your opinion about below CT Head image in a patient with altered mental status?

**Image 3.4** CT head

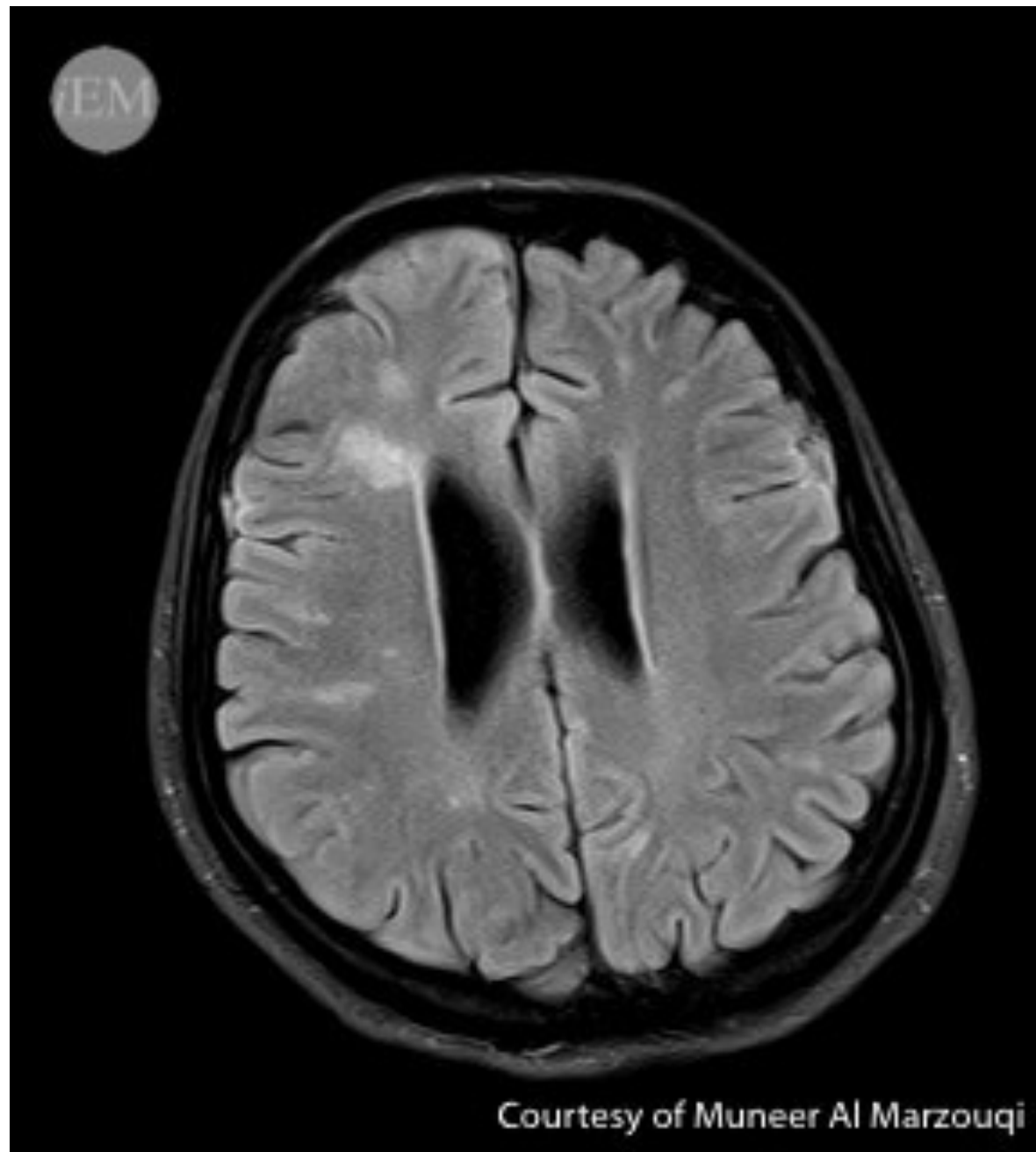


Case – Motor vehicle accident, head trauma, GCS 9

- Head MRI: In selected patients (e.g., ischemic stroke, sinus venous thrombosis)

What is your opinion about below MRI images in a patient with altered mental status?

**Image 3.5** MRI

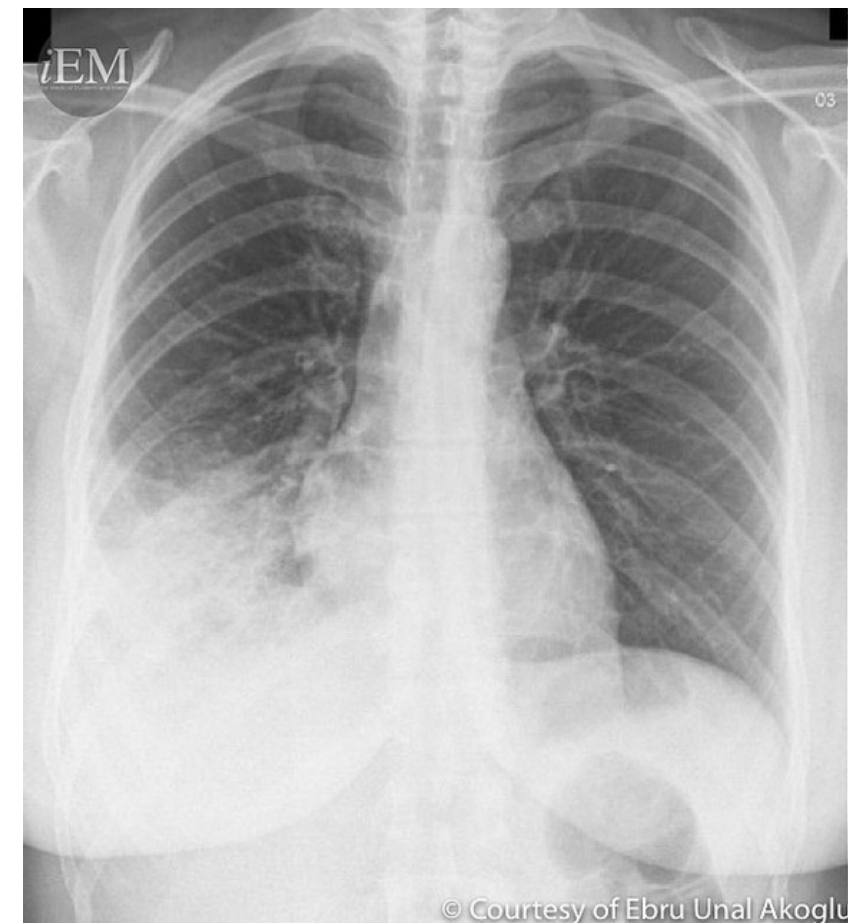


Case – 3 days history of fever, headache and gradual altered mental status

- EEG: If there is a suspicion for seizure, non-convulsive status epilepticus, etc.
- **Chest radiogram:** When indicated, for evaluating suspected cardiac and pulmonary conditions (e.g., pneumonia, pleural effusion)

What is your opinion about below chest x-ray in a patient with altered mental status?

**Image 3.6** Chest x-ray.



Case – 60 yo female, 7 days gradual history of dyspnea, cough, sputum and fever. Today, altered mental status.

## Emergency Treatment Options

### Initial Stabilization

As we mentioned above, “ABC” is the first step in the evaluation. Life-threatening conditions detected during these steps must be immediately intervened. After initial stabilization, a detailed examination must be performed and the underlying cause(s) must be investigated. Treatment strategies vary according to the underlying condition.

- If hypoglycemia is detected, dextrose (preferably D50W) should be administered intravenously.
- In undifferentiated comatose patients, A “coma cocktail” can be given. Oxygen is also in this category. A mnemonic called DONT stands for dextrose, oxygen, naloxone, and thiamine. The application of coma cocktail was changed over time. Today, we have bedside gluco-stick tests. Therefore, application of glucose mainly depends on the bedside glucose results. Similarly, we are using portable saturation devices, depending on the SatO2 level measured on the bedside, oxygen application decisions may change. Therefore, these drugs may not be routine blindly anymore. In addition, it is better to emphasize that flumazenil (a benzodiazepine antidote) is not in the coma cocktail, and should not be used routinely also.
- Empiric antibiotics must be initiated as soon as possible if sepsis or central nervous system infection is suspected.

Diagnostic tests and procedures such as performing LP must not delay antibiotics administration.

### Medications

- Dextrose: For hypoglycemic patients, 50 mL D50W.
- Thiamine: For Wernicke’s encephalopathy, 100 mg IV
- Naloxone: As an antidote for opioid overdose, 0.4 mg IV, can be repeated up to a total dose of 10 mg according to some resources.
- Glucocorticoids: When cerebral edema due to CNS mass lesions is detected in cranial CT
- Fluid resuscitation for indicated situations (e.g. hypotension, dehydration, DKA)

Specific antidote should be given in intoxications. Choose appropriate empiric antibiotics for the suspected source of infection and possible microorganisms. Broad-spectrum antibiotics are an option.

Intravenous insulin infusion for diabetic ketoacidosis or hyperosmolar hyperglycemic syndrome. Electrolyte imbalances must be corrected using appropriate fluid replacements.

### Procedures

- **LP:** Indicated if central nervous system infection or subarachnoid bleeding (with negative CT scan) is suspected.



## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

Advanced age is an independent risk factor for acutely altered mental status. Almost half of the patients presenting ED with altered mental status are elderly. Neurologic etiology is more common in the geriatric population and is the leading cause. Having multiple diseases and using multiple medications obscure the diagnostic process. When renal or hepatic functions are impaired, patients become more prone to drug intoxication without overdose. Moreover, drug interactions should be kept in mind, especially when a new medication is initiated.

In the pediatric population, symptoms and clinical findings may be very subtle and non-specific. Obtaining a detailed history, learning the child's baseline and shifts from that baseline, and repeated physical examinations are essential.

Etiology of altered mental status in the pediatric population differs from the one in the adults. In a child with an altered mental status, the mnemonic "VITAMINS" is helpful:

### Mnemonic VITAMINS

V-Vascular (e.g., AV malformations, vasculitis)

I-Infection (e.g., sepsis, meningitis, encephalitis)

T-Toxins (e.g., drugs, environmental toxins)

A-Accident/abuse (e.g., trauma with a suspicious history)

M-Metabolic (e.g., glycemic abnormalities, electrolyte imbalance)

I-Intussusception

N-Neoplasm

S-Seizure

In pregnant patients, in addition to the etiologies mentioned above, eclampsia must be kept in mind. Eclampsia, which is life-threatening both for the patient and fetus, is usually seen in the third trimester but can be seen during postpartum period as well. All pregnant patients with seizures or altered mental state must be investigated for eclampsia. Other conditions with increased risk during pregnancy are cerebral venous thrombosis, ischemic and hemorrhagic strokes.

## Disposition Decisions

The decision on discharging or admitting the patient depends on the underlying cause of mental status change and the clinical situation of the patient. The majority of the patients require admission, either to the ward or intensive care unit.

If the underlying cause is completely reversed and unlikely to re-occur, the patient reached his/her baseline mental status, vital signs are normal and stable, preparing a discharge plan may be considered.





References and Further Reading, click [here](#).

# Cardiac Arrest

---

by Abdel Noureldin and Falak Sayed

*A 23-year-old female was brought into the emergency department. Her frantic family members said they found her on the bathroom floor, not breathing, unresponsive, and with no pulse. EMS brought the patient to the ED.*

Spanish translation is available [here](#)



Audio is available [here](#)

## Introduction

Cardiac arrest is a condition that every emergency physician must be an expert in managing. The EM doctor will face it and have a love/hate relationship with it. You love it when the patient is resuscitated and breathing on his own; you can then tell the family their loved one is alive. You feel great because, after all, we are here to save lives, and it's the reason we joined this specialty. You hate it when your eyes are tearing up as you inform the parents of the newborn that he or she did not make it.

This illness is due to the lack of effective perfusion of the organs of the body 2nd to the abrupt failure of the heart to pump blood. Every year, over a quarter of a million lives are lost because of cardiopulmonary arrest, and most of these cases occur outside of the hospital. However, the mortality rate can be improved with the early and effective initiation of cardiopulmonary resuscitation (CPR) and advanced cardiopulmonary life support (ACLS).

## Cardiac Arrest Rhythms

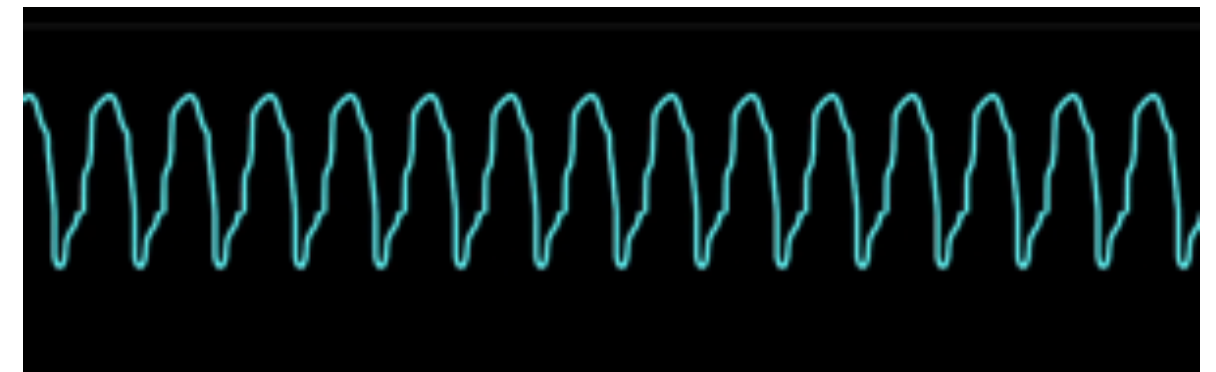
The pulseless cardiac arrest is caused by 4 different types of primary arrhythmias that consist of 2 shockable rhythms (ventricular tachycardia and ventricular fibrillation), and 2 non-shockable rhythms (pulseless electrical activity and asystole).

### Shockable rhythm

#### Ventricular Tachycardia

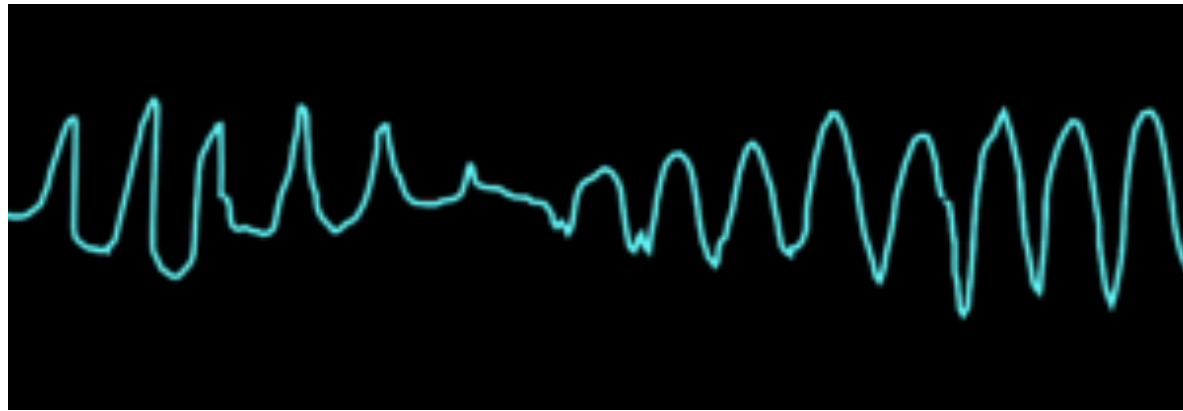
Ventricular tachycardia (VT) has 2 different types. The most common is the monomorphic (VT) and is defined as 3 or more consecutive ectopic ventricular beats (QRS complexes) of the same type.

**Image 3.7** Ventricular Tachycardia



The 2nd type is the polymorphic (Torsade's De pointes) that consists of ectopic ventricular beats (QRS complexes) of different types of morphology.

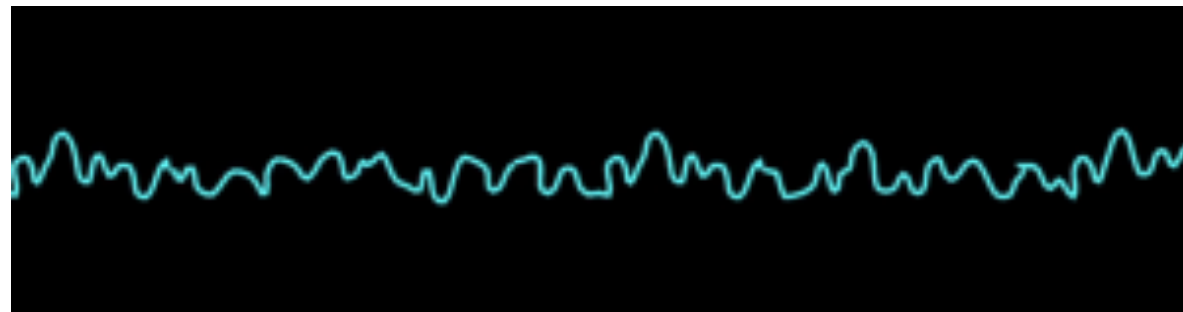
**Image 3.8** Torsade De Pointes



### Ventricular Fibrillation

Ventricular fibrillation is rapid and unorganized electrical impulses which makes the ventricles of the heart quiver while no pumping of the blood occurs.

**Image 3.9** Ventricular Fibrillation



## Non-shockable rhythms

### Pulseless Electrical Activity

Pulseless electrical activity (PEA) shows organized electrical rhythm on the electrocardiogram with no mechanical contractions of the heart muscle (no pulse). It is also called electromechanical dissociation.

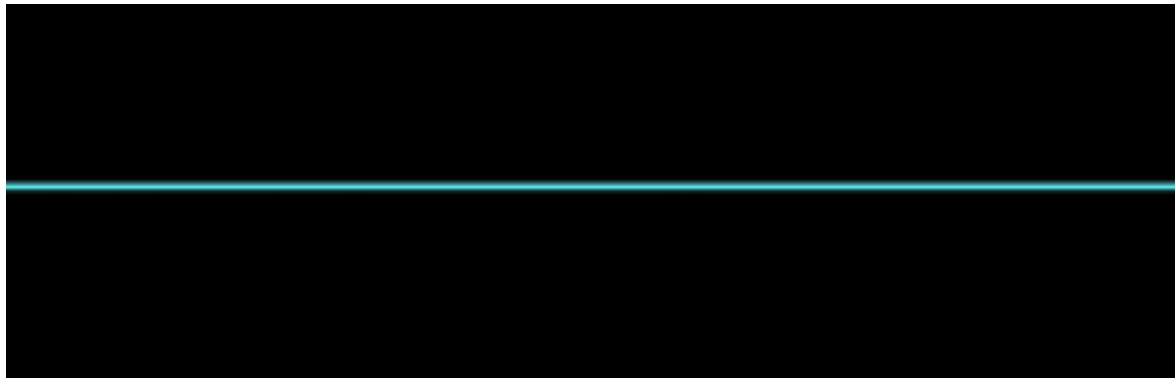
**Image 3.10** PEA



### Asystole

Asystole is defined as no electrical activity in the heart and no mechanical contraction of the heart muscle (no pulse). It is also called flat line or cardiac standstill.

**Image 3.11** Asystole



## Medications for Cardiac Arrest

There are only 3 emergency drugs you should now in any cardiac arrest patient. These are Epinephrine, Amiodarone, and Magnesium.

### Epinephrine

Concentration 1:10,000

Pediatric dosage 0.1 mL/Kg (20 kg child = 2 mL)

Adult dosage 10 mL or basically 1 mg

Frequency Every 2 cycles (3 to 5 minutes)

Indication All pulseless cardiac arrest rhythms

Mechanism : An agonist for the beta and alpha receptor which increases the perfusion pressure in the coronary and cerebral vessels

### Amiodarone

Pediatric dosage 5 mg/Kg (can be repeated up to 300 mg)

Adult dosage 300 mg (can be repeated at 150 mg)

Indication shockable rhythm (VT and VF)

Frequency 1st dose after the 3rd shock and repeat dose after the fourth defibrillation

Mechanism: Class III antiarrhythmic drugs.

### Magnesium

Pediatric dosage 25 to 50 mg/Kg (maximum 2 grams)

Adult dosage 1 to 2 grams

Indication Torsade de Pointes

Frequency Once when the diagnosis is made

Mechanism: Shorten the prolonged QT interval

## Imperative Concepts for The Team During Cardiac Arrest Management

### The compressors

👤 Must push hard (2 to 2.4 inches or 5 to 6 cm)

👤 Should push fast (100 -120/minute)

👤 Ought to allow the chest to recoil completely



- 📌 Rotate with another person every 2 minutes

## Airway

- 📌 Do not hyperventilate
- 📌 2 ventilations to 30 compressions while using ambo bag
- 📌 Give one breath every 6 seconds when the patient is intubated
- 📌 Use wave capnography to monitor CPR (CO<sub>2</sub> should be >10)
- 📌 If the advance airway is needed, use supraglottic devices or endotracheal intubation
- 📌 Confirm endotracheal intubation by wave capnography

## Shock delivery

- 📌 Biphasic – 200 Joules
- 📌 Monophasic – 360 Joules
- 📌 Make sure everyone is clear before you shock the patient
- 📌 Attach the patient to the monitor

## Drug therapy

- 📌 Start IV or IO
- 📌 Epinephrine: (1:10,000) 0.1ml/KG for pediatric and 10 ml for adult
- 📌 Amiodarone: 5mg/kg for pediatric and 300 mg for adult

- 📌 Amiodarone 2nd dose: up to 15 mg/kg (max-300) and 150mg adult

- 📌 Magnesium: 25 to 50 mg/KG- pediatric and 1 to 2 grams – adult

## Recorder

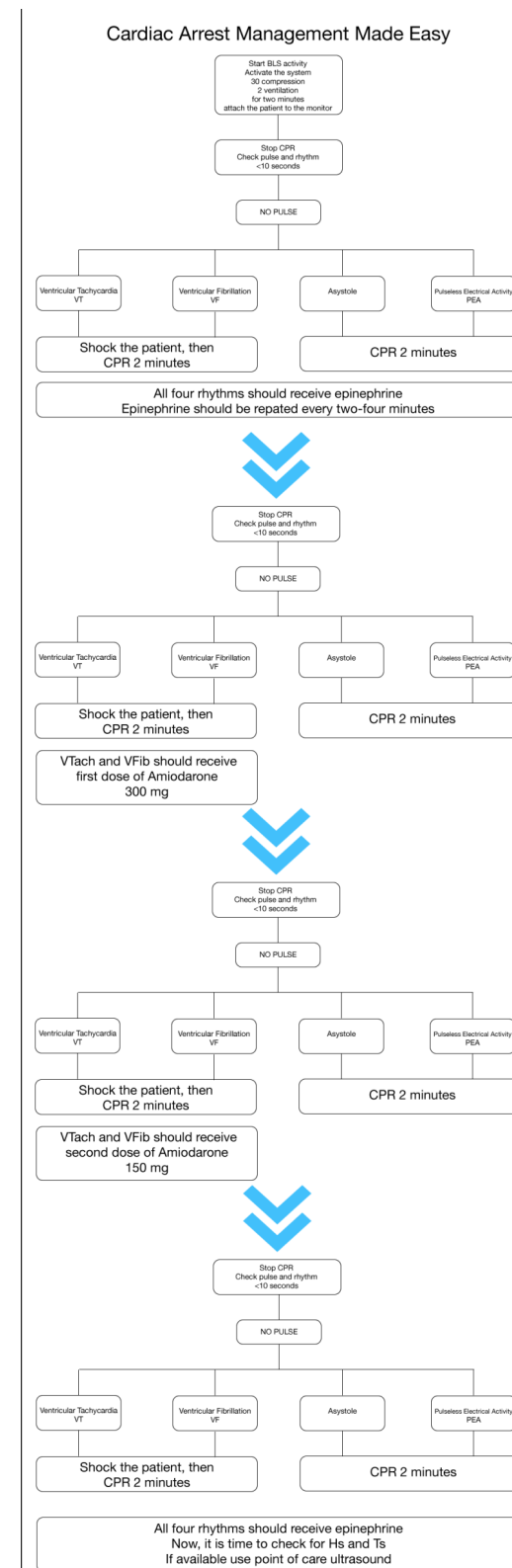
- 📌 Must record all the drugs given and the time it was given
- 📌 Inform the team members at the end of each cycle
- 📌 Keep track of the total time of resuscitation

## Team leader

- 📌 Must have mutual respect for all members of the team
- 📌 Look for ROSC (RETURN OF SPONTANEOUS CIRCULATION)
  - The sudden increase in the PETCO<sub>2</sub> (>40)
  - Return of pulse and pressure
- 📌 Make sure the interruption of chest compression is <10 seconds
- 📌 Remember the reversible causes (Hs and Ts)
  - Hypovolemia
  - Hypoxia
  - Hydrogen Ion (acidosis)

- Hypo-hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade
- Toxins
- Thrombosis (coronary and pulmonary)
- Trauma

**Diagram 3.1** Cardiac Arrest Management Made Easy



**If the patient developed a pulse at any time during resuscitation**

- Stop CPR
- Intubate the patient and secure the airway
- Start post resuscitation care
- Induced hypothermia
- Admit to ICU

**If the patient remained pulseless**

- Stop CPR after 20 minutes of resuscitation of the non-shockable rhythm or after 20 minutes from the last shock that was delivered.

**References and Further Reading, click [here](#).**

# Chest Pain

---

by Asaad S Shujaa

## Introduction

Chest pain is one of the most common symptoms presented in the emergency department (ED), and it is worrisome because the differential diagnosis widely range between non-emergent conditions and life-threatening conditions such as **acute coronary syndromes (ACS)**, **pulmonary embolism (PE)**, **aortic dissection**, **pericarditis with tamponade**, **pneumothorax**, and esophageal rupture. Chest pain caused by non-emergent conditions include esophageal reflux, peptic ulcer, **biliary colic**, muscle strain, costochondritis, pleurisy, **pneumonia** and non-specific chest wall pain.

It is important as emergency physicians to have an approach to chest pain that enables one to recognize life-threatening conditions from non-emergent conditions. This chapter aims to discuss how to approach a patient with chest pain.

Currently, we do not have data regarding how many patients visit the ED with chest pain in the Middle East; however, in the USA, approximately 6 million patients visit ED with chest pain, which accounts for almost 9% of all ED cases. This makes it the second most common complaint in ED visits.



Audio is available [here](#)

## General Approach to Patient with Chest Pain in Emergency Department

*“As a general rule, any chest pain is ischemic in origin until proven otherwise.”*

### Initial Approach

#### Airway, Breathing and Circulation (ABC) assessment

- Assessment of the airway by being able to talk without distress, no obvious upper airway obstruction such as tongue swelling, lip swelling, hoarseness, etc.
- Assessment of breathing by listening to the pulmonary sounds. Is it equal or wet (basal crackles indicate CHF)?
- Assessment of circulation by listening to heart sounds. Are there any S3,4 gallop rhythm (CHF) or new murmurs such as mitral regurgitation (papillary muscle dysfunction).
- Checking the pulses, capillary refill to understand the shock situation.

**Vital signs** should be assessed and repeated at regular intervals. For example, respiratory distress with low O<sub>2</sub> saturation may indicate pulmonary edema, plus low BP indicates cardiogenic

shock. Also, unequal BP in both arm or pulse deficient indicates aortic dissection.

#### The general appearance of the patient

- Looks sick or not sick or
- Patient in pain or not in pain

**Electrocardiogram (ECG):** To interpret myocardial ischemia, arrhythmias, pericarditis, and right ventricular strain findings for PE.

Any abnormality found in the initial approach may need immediate actions.

### History

#### What types of questions would you like to ask?

- Are you having discomfort, chest pain?
- How would you describe it?
- Where is it?
- Does it radiate anywhere?
- Frequency?
- Time of onset or acute worsening?
- Has there been any progression?

- Any aggravating/alleviating factors?
- Any associated symptom?
  - Diaphoresis, nausea, vomiting, cough, fever
- History of cardiopulmonary disease?
  - Risk factors for coronary disease such as hypertension, diabetes, high cholesterol, obesity, male, family history, smoker, sedentary, post-menopausal, previous history of ACS and family history of CAD.
  - Risk factors for pulmonary embolism such as travel history, oral contraceptive use etc. And risk factors for other critical diagnoses.

# Physical Examination

- Repeat assessment of the airway, breathing, and circulation with full examination steps.
- Assess abdomen for tenderness and pulsating mass
- Look for swelling in legs (lower limb edema), calf tenderness (deep vein thrombosis).

**Table 3.9** History taking for chest pain

iEM Table 1: Chest Pain History		
Type of Chest Pain	Pressure, tightness, or heaviness, sharp, tearing or ripping	ACS represents with pressure, tightness or heaviness type 1 pain. Sharp pain is generally from problems involving pleura such as pneumothorax. PE also represent with sharp or dull pain. Tearing pain generally, refer to aortic dissection.
Location	Central, left, or right	All critical diagnoses can be located in one of those. Location not helpful to differentiate the diseases.
Timing	Gradual or sudden onset	Pneumothorax, PE generally start sudden onset. Pneumonia, pericarditis represents with gradual pain.
Severity	Scale of 0-10	It may not be a relevant item to decide the severity of diagnosis because of it varies between patients because it depends on their pain threshold. However, in general, PE, Aortic dissection, MI, pericarditis, esophageal rupture represents with severe pain.
Radiation	Back, neck, arm and jaw	ACS prone to radiate jaw, neck, arm, shoulder. But, pneumothorax also radiate to the neck, shoulder area. Aortic dissection represents with back pain along with chest pain.
Provocations	What makes it worse or better?	Physical activity, acute increase stress may trigger ACS pain. Deep breath, positional changes may worsen pneumothorax pain. Positional changes are also important for pericarditis. Lying down increases the pain while sitting or leaning forward decrease it.
Associated symptoms	SOB, sweating, nausea and vomiting	Pneumothorax, pneumonia, PE are associated with the SOB. ACS, pneumonia, esophageal rupture may present with sweating.

Produced by Asaad Suliman Shujaa (Ed. AAC)



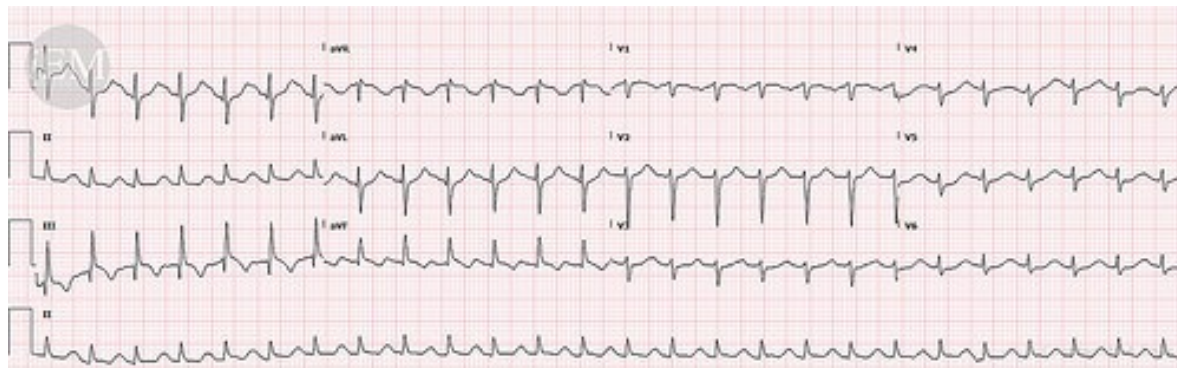
## Bedside test

### ECG

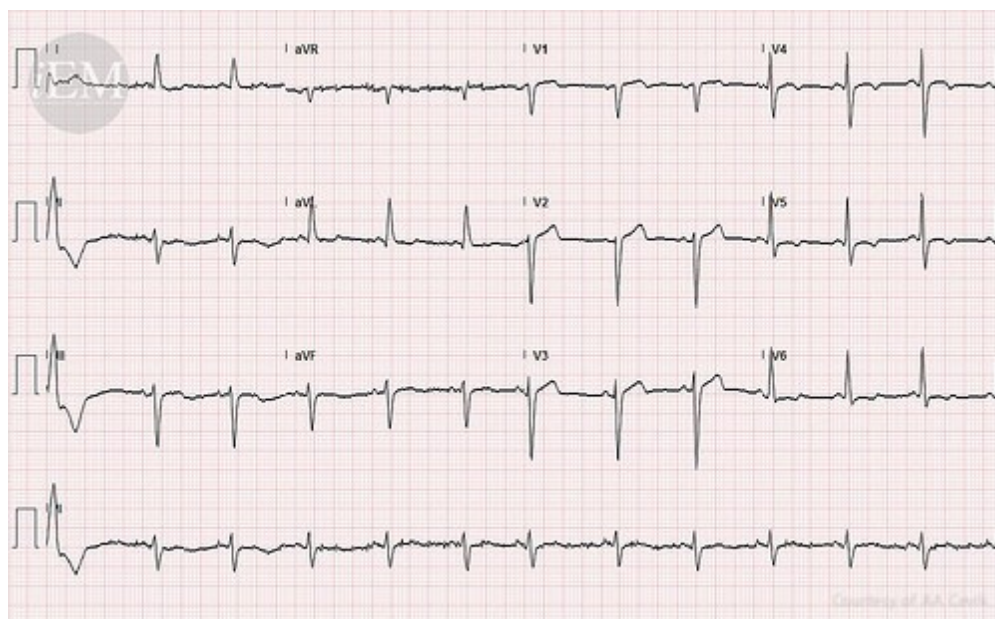
ECG is the main bedside test for any chest pain patient.

What is your opinion about below ECGs in patients with different type of chest pain?

**Image 3.12** Case – 54 yo female presented with 3 days history of right side chest pain (pleuritic).



**Image 3.13** Case – 46 yo male presented with central chest pain. He has nausea and diaphoresis.



- 12 lead ECG for myocardial infarction and 15 lead ECG for posterior myocardial infarction
  - Any ST elevation in 2 contiguous leads should be evaluated as S.T. Elevation M.I. However, please do remember, there are many other problems can elevate S.T. segment.
  - Any other changes such as ST depression, T inversion and Q wave should be evaluated
- ECG is more useful as 'rule in' than 'rule out.'
- In Acute Myocardial Infarction ECG has 50% sensitivity, 90% specificity.
- 12 lead ECG for PE may show S1 Q3 T3 sign (prominent S wave in the lead I, Q wave and inverted T wave in the lead III). It is a sign of acute right ventricular strain (acute pressure and volume overload of the right ventricle because of pulmonary hypertension). Other ECG findings noted during the acute phase of a PE include new right bundle branch block (complete or incomplete), rightward shift of the QRS axis, ST-segment elevation in V1 and aVR, generalized low amplitude QRS complexes, atrial premature contractions, sinus tachycardia, atrial fibrillation/flutter, and T wave inversions in leads V1-V4.
- The ECG is often abnormal in PE, but findings are neither sensitive nor specific for the diagnosis of PE. The greatest utility

of the ECG in a patient with suspected PE is ruling out other life-threatening diagnoses such as acute myocardial infarction.

- Some aortic dissection cases may also show ST-segment elevation as in acute myocardial infarction.
- ECG may also help to diagnose pericarditis, especially chest pain patients with fever.

## Laboratory tests

### Cardiac markers

- Troponin I or T rise within 3-6 hours and then remain elevated for about one week
- Serial testing improves sensitivity
- In acute coronary syndrome suspicion, an increased Troponin is a marker for increased risk of AMI and death
- However, cardiac enzymes do not diagnose cardiac ischemia

### D-dimer

- Only use is in a low-risk patient
- A negative test makes PE very unlikely
- A slightly positive test is a positive test

## Others

Complete blood count, ESR, C reactive protein, blood culture, and lactate may help to rule out some infections such as pericarditis or mediastinitis because of esophageal rupture. But, their value in the acute setting is questionable.

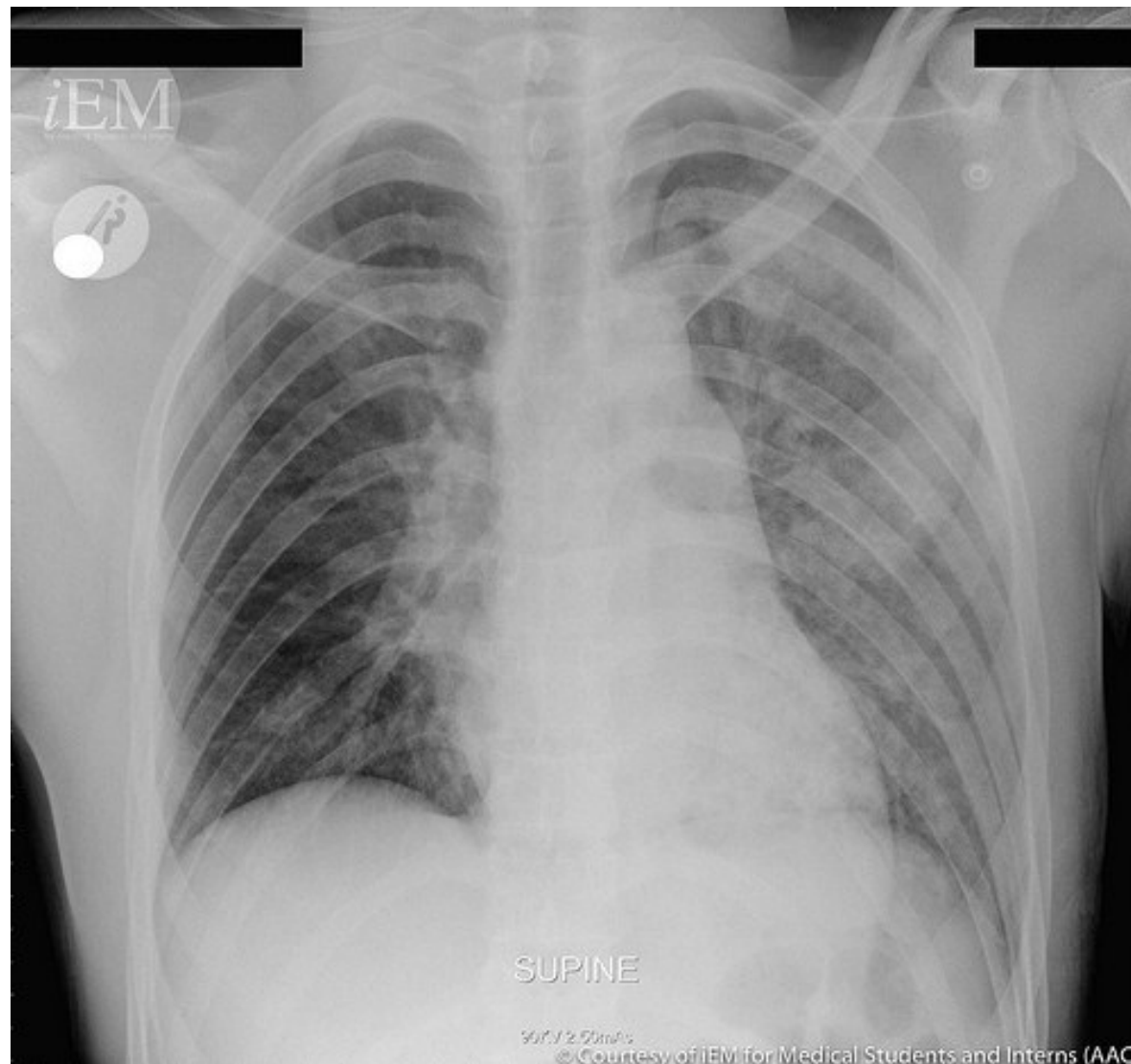
## Imaging modalities

### Chest X-Ray

- To look for heart failure and evaluate for other cause of chest pain such as Aortic Dissection, pneumothorax, pneumonia etc.
- Widened mediastinum, abnormal aortic knob, pleural effusions for aortic dissection. These findings are not sensitive for the aortic dissection. Only 25% of the patients have wide mediastinum.
- Esophageal rupture signs in chest X-ray; Hydropneumothorax, Pneumothorax, Pneumomediastinum, Subcutaneous Emphysema, Mediastinal widening without emphysema, Subdiaphragmatic air and Pleural Effusion.

What is your opinion about below chest x-ray in a patient with chest pain?

**Image 3.14** Case – 58 yo male presented with 1 day history of sudden onset left side chest pain radiating to left shoulder.



### Bedside ultrasound

- **RUSH** protocol evaluates aorta and pericardial space to rule out tamponade ([video](#))
- Consider Doppler ultrasound to see deep vein thrombosis in legs ([video](#))

### CT scan

- CT with contrast shows large, central emboli, it is also very sensitive for aortic dissection.
- In the suspicion of esophageal rupture, contrast-enhanced CT scan of the chest should be obtained if it is not possible to obtain a contrast esophagogram, if the esophagogram was negative, despite a high clinical suspicion, or if seeking to evaluate for a more likely alternative diagnosis. Perforation may be suggested by mediastinal air, extravasated luminal contrast, peri-esophageal fluid collections, pleural effusions, or actual communication of an air-filled esophagus with an adjacent mediastinal air-fluid collection. Definitive esophageal communication with outside structures is often difficult to visualize.
- The pulmonary angiogram is the gold standard for PE and aortic dissection but carries a risk of contrast-induced nephrotoxicity and anaphylactic contrast reaction.

### V/Q scan

It is very sensitive but not specific for patients with suspected PE.

Depending on your history, physical exam and bedside investigations as well as laboratory and imaging results, the focus should be given to rule out myocardial ischemia or infarction, pulmonary embolus, pneumothorax, pericarditis with tamponade, aortic dissection, and esophageal rupture. Each of this specific

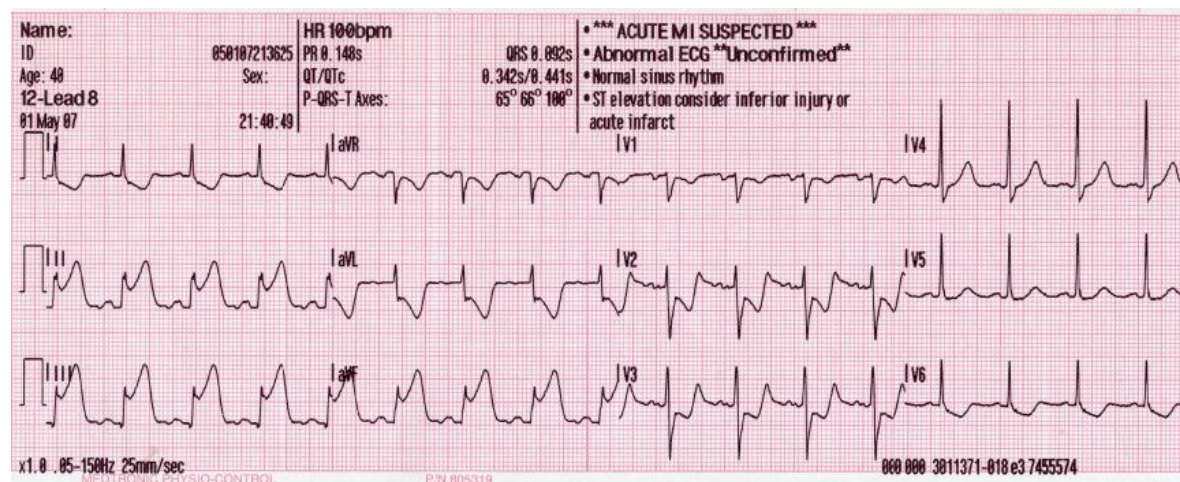


disease entities has various risk stratification methods, treatment options, and dispositions. Now, it is time to look to some cases and discuss more specific management in the ED.

## Case 1

A 46-year-old male with a history of diabetes mellitus, hypertension, and coronary artery disease presents to the ED. He is a smoker. He complains of chest tightness and heaviness. The symptom started gradually 3 hours ago and lasts 20 minutes when he was watching TV. The pain scale was 5/10, radiated to his jaw. The pain is associated with nausea and sweating. He took Nitroglycerin spray, and the pain was relieved. The pain started again before he reached the ED. The pain scale is 10/10. The initial assessment at triage: ABC intact, BP: 140/80, HR: 110 RR: 24, O2Sat: 98% on room air, Temperature: 37.3, Random Blood Sugar: normal.

Image 3.15 ECG case 1



## Case 1 – Critical Bedside Actions and General Approach

- 📌 Place the patient in a monitored bed, make sure security chamber established (monitor, IVs, oxygen, etc.)
- 📌 ABC intact
- 📌 Vitally stable except he has tachycardia (HR: 110)
- 📌 Quick History and Physical Examination as described in the text. Chest exam: Equal air entry, no wheeze or crackles
- 📌 CVS exam: S1+S2 no additional sound, no murmur, JVP was normal
- 📌 No lower limb edema, pulses for four limb present and equal
- 📌 12 lead ECG shows inferior ST-elevation myocardial infarction
- 📌 Consult cardiologist as soon as possible
- 📌 Patient in pain needs analgesia
- 📌 Aspirin 300 mg was given by EMS

## Case 1 – Differential Diagnoses

There are six life-threatening differential diagnoses for any chest pain patients. These consist of:

1. Myocardial ischemia or infarction (MI)
2. Pulmonary embolus (PE)
3. Pneumothorax
4. Pericarditis with tamponade
5. Aortic dissection
6. Esophageal rupture

## Case 1 – History and Physical Examination Hints

- The chest pain is typical angina pain (heaviness radiating to jaw associated with nausea and sweating), the pain is not sharp such as in PE or tearing like in aortic dissection
- The patient has cardiac risk factors (DM, HTN, CAD, Smoker, and MI 1 year ago)
- No PE risk factors

- The history does not suggest any past esophageal rupture
- Physical exam not lead to cardiogenic shock or pulmonary edema
- No sign of pneumothorax in the exam
- Pulses all equal for four limbs and no inequality in BP in both arms, which does not go with aortic dissection
- ECG suggested Inferior MI, no sign of pericarditis in ECG

## Case 1 – Emergency Diagnostic Tests and Interpretation

- ECG suggested Inferior MI, no sign of pericarditis in ECG
- Portable CXR: normal which rules out pneumothorax and aortic dissection (no wide mediastinum)
- Troponin I is high, which suggests Myocardia Ischemia
- In bedside echocardiography, there is hypokinetic in the inferior wall and no sign of cardiac tamponade

## Case 1 – Emergency Treatment

- Aspirin should be given immediately
  - Great benefit, little risk
  - Give the minimum of 182 mg
- Rapid decisions on reperfusion
  - Based on ECG only (PCI vs. Fibrinolysis)
- Antiplatelet options:
  - Heparin (LMWH versus unfractionated)
  - Clopidogrel
- Symptomatic / pain control
  - GTN Vasodilator also reduces preload
  - Can give SL or IV
  - Morphine for pain control and reduce anxiety and stress
- Secondary prevention



- B-Blocker, statins and ACE inhibitor

## Case 1 – Disposition Decision

Assess the risk stratification by using  
**TIMI score**

## Case 1 – Admission criteria

- Establish risk level using the TIMI scoring system
- Moderate risk: Admit for further evaluation; add beta blockers, ACE inhibitors. Follow cardiac enzyme levels. If MI ruled out, exercise stress test before discharge
- High Risk: Admit for cardiac catheterization

## Case 1 – Discharge criteria

- Low-risk TIMI score: May be discharged after symptom control and follow up with cardiologist outpatient for the stress test and lipid profile test

## Case 1 – Referral

- Cardiology

## Case 2

A 30-year-old male had an open reduction and internal fixation (ORIF) of right ankle fracture 2 weeks ago. C/O sudden onset of chest pain today. He has pleuritic sharp chest pain associated with short breath, increased during inspiration.

## Initial assessment at triage

- ABC intact
- Vital signs
  - BP 120/80
  - Pulse 120
  - RR 40
  - O2 sat 88% on room air
  - T 36.5
- 12 ECG shows sinus tachycardia, T inversion V2,3 and 4, deep S lead I and Q and T inversion in the lead III, ST elevation V1 and V4R suggested pulmonary embolism

## Case 2 – Critical Bedside Actions and General Approach

- O2 Supply and monitor bed
- ABC intact
- Vitally stable except he is tachycardia (HR 120)
- The quick history that suggested the patient had a major surgery 2 weeks ago and was immobilized 2 weeks.
- Physical examination shows
  - Chest exam: Equal air entry, no wheeze or crackles
  - CVS exam: S1+S2 no additional sound, no murmur, JVP was normal
  - There is calf swelling in right site of surgery, pulses for 4 limbs present and equal
- To do 12 lead ECG shows sinus tachycardia, T inversion V2,3 and 4, deep S lead I and Q and T inversion in the lead III, ST elevation V1 and V4R suggested pulmonary embolism

- Patient in pain need analgesia

## Case 2 – Differential Diagnoses

1. Pulmonary embolus (PE)
2. Myocardial ischemia or infarction (MI)
3. Pneumothorax
4. Pericarditis with Tamponade
5. Aortic dissection
6. Esophageal rupture

## Case 2 – History and Physical Examination Hints

- The chest Pain is atypical angina pain (sharp, pleuritic chest pain increased by inspiration and associated with shortness of breath, no radiation), the pain is not angina pain OR no tearing pain as in aortic dissection
- There are PE risk factors (major surgery, immobilization 2 weeks)
- The history does not suggest any previous Esophageal rupture

- Physical exam not lead to pneumonia no crackles in chest exam
- No sign of pneumothorax in the exam
- Pulses all equal for four limbs and no inequality in BP in both arms, which does not go with aortic dissection
- ECG suggested PE, no sign of pericarditis in ECG

## Case 2 – Emergency Diagnostic Tests and Interpretation

- ECG suggested Pulmonary embolism, no sign of pericarditis in ECG
- Portable CXR: normal which rules out pneumothorax and aortic dissection (no wide mediastinum)
- D- Dimer is high
- Cardiac enzymes are negative
- Bedside echocardiography there is signs of right ventricle enlargement and strain and no sign of cardiac tamponade

## Case 2 – Emergency Treatment

- Heparin (Will limit propagation but does not dissolve clot)
  - Unfractionated: 80 u/kg bolus, 18 h/kg/hr.
  - Fractionated (Lovenox): 1 mg/kg SC BID
- Fibrinolysis
  - Consider with large if the patient is unstable
  - No study has shown a survival benefit, but it is very difficult to study.
  - Alteplase 50–100 mg infused over 2–6 hrs (bolus in severe shock)

## Case 2 – Disposition

If there is suspicious of PE, we need to do pre-test probability; there are multiple systems for doing this. Most widespread and validated is Well's score

There is a difference in Well's score for PE & DVT

## PE – Well’s criteria

- 3 points for:
  - PE ‘most likely diagnosis
  - Signs and symptoms suggesting DVT
- 1.5 points for:
  - PR>100,
  - history (PE/DVT),
  - immobilization in 2 weeks
- 1 point for:
  - Hemoptysis or malignancy

## Risk Stratification

- <2 low risk (10%), D-Dimer is good to rule out PE
- 2-6 medium risk (25%), Spiral CT chest with contrast to rule out PE
- >6 high (50%), start anticoagulation(LWMH) and Spiral CT chest with contrast

## Case 2 – Referral

- ICU
  - Unstable Patient, massive PE, Bilateral PE
- Medical Ward
  - Stable patient with Small PE

## Case 3

A 60-year-old male patient presented to the ED with sudden onset central chest pain, described as ripping his chest and radiating to the back, no associated symptoms and patient, previous history with HTN, CAD, and smoker. Initial assessment by EMS was ABC intact. Vitals were BP 190/95 Right arm, Pulse 110, RR 20 , T 37 , O2sat 98%.

## Case 3 – Critical Bedside Actions and General Approach

- O2 Supply and monitor bed
- ABC intact
- Vitally stable except he is high BP 185/85 mmHg on the right arm and

200/100 on the left arm, tachycardia (HR 110)

- Quick history which suggested sudden onset central chest pain, described as ripping his chest and radiating to the back, no associated symptoms.
- Physical examination shows:
  - Chest exam: Equal air entry, no wheeze or crackles
  - CVS exam: S1+S2, a grade 2/6 systolic murmur, and a soft decrescendo diastolic murmur are heard at the second right intercostal space. JVP was normal
  - There is radial to radial pulsation delay
  - There are abdominal and bilateral femoral bruits, with absent distal pulses.
- 12 lead ECG shows no ST, T wave changes, no sign of MI

- Portable CXR shows wide mediastinum, no sign of CHF, pneumothorax or pneumonia
- Patient in pain need analgesia

### Case 3 – Differential Diagnoses

1. Aortic dissection
2. Myocardial ischemia or infarction (MI)
3. Pulmonary embolus (PE)
4. Pneumothorax
5. Pericarditis with Tamponade
6. Esophageal rupture

### Case 3 – History and Physical Examination Hints

- The chest Pain is sudden onset central ripping chest pain radiating to back as in aortic dissection; the pain is not angina pain.
- There are risk factors: HTN, CAD, smoker, and age
- The history does not suggest any previous esophageal rupture

- Physical exam not lead to pneumonia, no crackles in chest exam
- No sign of pneumothorax in the exam
- Pulses delay in radio –radio pulsation and different BP in both arm and abdominal and bilateral femoral bruits, with absent distal pulses with going with aortic dissection
- ECG no sign of ischemic changes, no sign of pericarditis in ECG
- Patient in Pain need analgesia

### Case 3 – Emergency Diagnostic Tests and Interpretation

- 12 lead ECG shows no ST, T wave changes, no sign of MI
- Portable CXR shows wide mediastinum, no sign of CHF, pneumothorax or pneumonia
- The cardiac enzyme was negative rule out MI
- D-Dimer was negative

• Bedside Echo has no sign of tamponade

- CT scan is the most accurate and fastest option

### Case 3 – Emergency Treatment

- Involve Cardio-Thoracic surgery as soon as possible.
- Control the blood pressure
- SBP goal is 120-130 mmHg
- Beta blockers are first-line agents (Labetalol and Esmolol), they control blood pressure and heart rate
- Depending on the patient's vitals you can add vasodilators such as nitroprusside

### Case 3 – Disposition

- Patients should be admitted to ICU,
- Emergency surgery is needed for ascending dissections
- If dissection is only descending, management is only supportive.

## Case 4

A 55-year-old alcoholic with persistent vomiting presents with sudden onset of Chest Pain followed by hematemesis. The chest pain is sudden onset, sharp in nature, radiating to the back. It is associated with shortness of breath for 3 hours. Past medical history: DM, HTN, alcoholic, and smoker. Vitals: BP 120/80 equal bilateral arm, pulse 90 regular and equal on four limbs, no pulse deficit, RR 40, T 38, O2sat 96% on room air.

### Case 4 – Critical Bedside Actions and General Approach

- O2 Supply and monitor bed
- ABC intact
- Vitally stable except he is febrile (T 38)
- The quick history which suggested the sudden onset of Chest Pain followed by hematemesis. The chest pain is sudden onset, sharp in nature radiating to the back; it is associated with shortness of breath for 3 hours.
- Physical examination shows

- Chest exam: decrease air entry in the left side, and there is subcutaneous emphysema in the left side of the chest
- CVS exam: S1+S2. No additional sound, JVP was normal, pulses equal in four limbs
- 12 lead ECG shows no ST, T wave changes, or ischemic changes
- Portable CXR shows left pleural effusion and pneumomediastinum and normal width of the mediastinum.

### Case 4 – Differential Diagnoses

1. Esophageal rupture
2. Aortic dissection
3. Myocardial ischemia or infarction (MI)
4. Pulmonary embolus (PE)
5. Pneumothorax
6. Pericarditis with Tamponade

### Case 4 – History and Physical Examination Hints

- The chest pain is sudden onset followed by hematemesis. The chest pain is sharp in nature radiating to the back; it is associated with shortness of breath for 3 hours. A history of repeated vomiting and associated with short of breath and vomiting blood (hematemesis).
- There is Risk factors, HTN, CAD, smoking, and alcohol use
- There is strong history suggested of Esophageal rupture
- Physical exam shows decreased air entry in the left side, and there is subcutaneous emphysema in the left side of the chest
- No sign of pneumothorax in the exam
- ECG no sign of ischemic changes, no sign of pericarditis in ECG



## Case 4 – Emergency Diagnostic Tests and Interpretation

- 12 lead ECG shows no ST, T wave changes, no sign of MI
- Portable CXR shows left pleural effusion and right pneumomediastinum and normal width of the mediastinum. No sign of pneumothorax, no sign of CHF, no sign of pneumonia
- Cardiac enzymes were negative, which rule out MI
- D-Dimer was negative
- Bedside Echo: no sign of tamponade

## Case 4 – Emergency Treatment and Disposition

- Nothing by mouth, NPO
- Broad-spectrum antibiotics – No randomized clinical trials exist for antibiotics and esophageal perforation; however, empiric coverage for anaerobic and both gram-negative and gram-positive aerobes should be

initiated when the initial diagnosis is suspected.

- Parenteral nutritional support
- Nasogastric suction – This should be maintained until there is evidence to indicate that the esophageal perforation has healed, is smaller or is unchanged
- Narcotic analgesics
- Admission to a medical or surgical intensive care unit (ICU)
- Outcome: survival 65-90%, poor survival with delayed diagnosis >48hrs

**References and Further Reading**, click [here](#).

# A Child With Fever

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by Jabeen Fayyaz

## Case Presentation

*A 2-month-old female child was brought in with a history of cough and fever for 2 days. As per mother, the fever was high grade, documented as 38.5°C with an inability to drink for the last 4 hours. There was history of an episode of cyanosis at home with coughing an hour ago. On examination, the child was looking dull and lethargic. Her vital signs were: Temp 39°C, HR 170/ min, RR 65/ min, SPO2 89% in room air, BP 75/50mm of Hg, and Capillary refill time 4 sec. Chest on auscultation has bilateral crepitation. The child was taken immediately in the resuscitation area and was put on high flow oxygen. The blood work up and CX-ray ordered showed right middle zone consolidation. IV antibiotic, Cefotaxime was administered. The child was kept on IV fluids and cardiac monitoring. The child was admitted to the high dependency unit.*

## Overview

Fever is one of the most common reasons for the Pediatric Emergency Department (PED) visits. It accounts for almost 10% to 25% of PED visits annually. Febrile illness in children is caused **mostly by viral infections**, but a significant proportion, especially in children who are less than 3 months old, are caused by serious bacterial infection (SBI). As an ED physician, the goal is to identify this population at risk and to promptly manage them.

SBI has been reported to affect 6-10 % of infants who are younger than 3 months and 5-7% of children who are between 3-36 months of age. Therefore, you should always be very careful when evaluating a child with fever under 3 years old. The infant's immune system is relatively immature during the first 2 to 3 months of life. This puts them in a very high risk group.

SBI can even be found in the presence of viral infection concomitantly, 5% of patients with confirmed viral sources having urinary tract infections or other SBIs. Infants and children presenting with a fever and signs of a viral illness should have investigations to confirm the viral etiology, but should also be assessed for other sources of bacterial infections. Details of this approach can be found in [Policy Clinical Guideline](#). Children with an apparent focus or are sick looking are easy to manage. However, it is very challenging and many gray zones in managing the well-appearing infants and children with febrile illness without any source in the chaotic ED environment. Febrile illness in

children results in significant parental anxiety. Management decisions about febrile children are further complicated by the fact that parents and physicians weigh the risks and costs differently.

In a study (Byington 2004), common sources of bacterial infection in children less than 90 days were found UTI, bacteremia, soft tissue infection, meningitis, and pneumonia.

**Fever** is defined as temperature  $\geq 38^{\circ}\text{C}$  measured rectally or tympanic/axillary temperature of approximately  $37.5^{\circ}\text{C}$ . If parents state that fever is documented at home by a thermometer, it should be considered as fever recorded in the ED and should be evaluated in the same manner. Another important consideration mainly in neonates is hypothermia. Neonates may respond to SBI with **hypothermia** rather than hyperthermia, so they need to be evaluated carefully for any other sign of toxicity.

Temperature in children can be measured at the axilla, rectally, orally or via the ear (tympanic). Younger children (<5 years old) cannot manage the glass thermometers because it can break easily. Therefore, this method is not recommended for this age group. To check the temperature in newborns and young children, axillary measurement is an acceptable method. However, children under 2 years of age may need confirmation with a rectal temperature. **Rectal temperature** is considered the gold standard. Bundling a young child may increase the skin temperature but not the core temperature. It should also be

considered in neonates and children less than 2 years of age where other methods are not reliable.

**Table 3.10** Normal temperature ranges in children measured by different method

MEASUREMENT METHOD	NORMAL TEMPERATURE RANGE
Rectal	36.6°C to 38°C (97.9°F to 100.4°F)
Ear	35.8°C to 38°C (96.4°F to 100.4°F)
Oral	35.5°C to 37.5°C (95.9°F to 99.5°F)
Axillary	34.7°C to 37.3°C (94.5°F to 99.1°F)

Temperature measurement in pediatrics - MicroLife USA, [http://www.microlifeusa.com/pdfs/therm/taking\\_an\\_infants\\_temp.pdf](http://www.microlifeusa.com/pdfs/therm/taking_an_infants_temp.pdf) (accessed June 27, 2016).

Studies have shown a good correlation between the tympanic temperature and rectal temperature, especially in children more than 2 years of age. On the contrary, axillary temperatures have a lower correlation. If there is any doubt about a child's temperature, rectal temperature measurement should be considered for confirmation. Rectal temperature should be **avoided** in neutropenic and immunocompromised children

**Table 3.11** Recommended methods to measure temperature by age

AGE	OPTIONS	RECOMMENDED TECHNIQUE
Birth -2 years	First Choice	Rectal (for exact temperature)
	Second Choice	Axillary (to check for fever)
Between 2 and 5 years	First Choice	Rectal
	Second Choice	Tympanic
	Third Choice	Axillary
Older than 5 years	First Choice	Oral
	Second Choice	Tympanic
	Third Choice	Axillary

provided by author.

## History and Physical Examination Hints

The detailed history and physical examination are the most vital in the assessment of the febrile child. It is critical to pay attention to the history provided by parents for documented fever at home as studies have shown it is moderately accurate; further evaluation should always be carried out because a subjective fever at home may be the only indicator of a possibly serious bacterial infection in a child who is afebrile in the ED.

Focused history on fever characteristics should be asked, as it may provide useful clues. There is an increase in the rate of pneumococcal bacteremia with a rise in temperature, especially in young children. Studies suggest that the incidence of SBI is higher in patients who have higher temperatures. The duration of the fever at the time of ED presentation does not help to predict occult bacteremia. The response to antipyretic medications does not predict bacterial or viral infection. Other important data to be considered include associated signs and symptoms, underlying medical conditions, exposure to ill contacts, and immunization status.

In the exam, evaluate the quality of the cry? High pitched, or weak in effort? Does the child appear fearful of the doctor, nurse? It is normal to see healthy young children's fear of strangers. Therefore, if you expose to a child who lies on the exam bed and not interacting things around him or not showing his/her fear, then you need to think about more serious illness on those.

Examination of skin is very important. So, skin color, cyanosis or jaundice, rashes should be evaluated. Although the skin may give a clue about the degree of hydration, tears during crying, moisture on the oral mucosa/lips and tongue should be checked. For the neonate, "gentle" palpation of the anterior fontanelle indicates current the fluid status. If the fontanelle is sunken, this shows hypovolemia/dehydration.

An assessment of the child's overall appearance is critical. Although there is an imperfect correlation between physical examination findings and serious bacterial illness, ill-appearing children are more likely than well-appearing children to have serious bacterial infection, and most well-appearing children do not have serious bacterial infection.

Toxic appearance includes lethargy with poor perfusion (delayed capillary refill, cold hands and feet, pale or mottled skin) or cyanosis or respiratory distress findings. Grunting is considered one of the most important signs to identify a sick child and may indicate an impending respiratory failure.



**Table 3.12** Assessment of Clinical Condition in Children on Physical Examination

	WELL	UNWELL	TOXIC
<b>Alertness/ Activity</b>	Strong cry or not crying / smiles / stay awake/ normal response to social cues	Drowsy / decreased activity / poor smile/response to social cues/ irritable	Wakes only with prolonged stimulation/ unable to arouse/ weak cry / high pitched cry/ continuous cry/ bulging fontanelle / grunting
<b>Breathing</b>	Normal work of breathing	Nasal flaring	Chest indrawing / RR more than age specific rates/ grunting
<b>Color / Circulation</b>	Normal lips, skin, tongue color	Pale per caregiver	Pale / mottled / blue / ashen
<b>Fluid/ Urine output</b>	Normal skin and eyes/ moist mucus membranes	Poor feeding in infants / dry mucus membranes / reduced urine output	Reduced skin turgor / bilious vomiting
<b>Others</b>		New lump >2 cm	Appears very unwell to health care professional

*provided by author*

The physical examination may reveal focal infection, and if so the need for additional testing decreases. For example, the children who have clinically obvious viral illness such as croup, chicken pox have lower rates of bacteremia than the children who have no obvious infection source. **Except for neonates and young infants**, if a child has a nontoxic appearance, a more selective approach can be undertaken. When a child has an identifiable cause, the treatment and disposition should generally be tailored to this specific infection.

NICE **green light system**.

## Emergency Treatment Options

Airway, breathing, circulation (ABC) is the priority for all patients. Supplemental oxygen or advance airway measure can be necessary. Open intravenous access to draw blood samples, fluid infusion, and medications. Monitor the patients' vitals. Early treatment of fever is important. This gives the patient comfort as well as optimal physical examination condition for the physician.

Acetaminophen and ibuprofen both can be used. They can overlap during the treatment period to control fever. Some studies favor acetaminophen because of its fast effect. Other studies found that combination of acetaminophen and ibuprofen is very effective regime. Recommended doses are acetaminophen 15 mg/kg and ibuprofen 10 mg/kg.

## Empiric antibiotic regimes should also be considered

- 👤 Age 0-28 days: ampicillin + gentamicin or a third-generation cephalosporin
- 👤 Age 29-56 days: Ill appearing children can receive same regimen above. The children who can discharge home do not need empiric antibiotics.
- 👤 Ceftriaxone is a preferred agent by some clinicians before ED discharge.
- 👤 Age 2-24 months: Empiric antibiotic therapy is not indicated for well-appearing children if there is no defined bacterial source and will be managed as outpatients.

Finding venous access, waiting the lab results and availability of the antibiotics are couple obstacles to apply timely antibiotics to children with fever.

## Disposition Decisions

Toxic appearance, need for monitoring, need for fluid treatment, poor social condition, follow up chance for the next

day with the primary physician are factors affecting admission decisions. However, admission is warranted for febrile infants 28-56 days old regardless of the above factors.

If the patient meets all of the following low-risk criteria, they may be discharged home.

- Full-term birth
- Not hospitalized longer than the mother
- No toxic appearing
- Not received antibiotic within 48 h
- No dehydration
- No lethargy
- No irritability
- No wheezing
- No infections in the ear, skin, soft tissue, skeletal
- No focal infection source
- No hyperbilirubinemia

• No underlying or chronic illness

- No previous admissions
- CSF – WBC < 8/hpf
- WBC – 500-15000/mm<sup>3</sup>, PMNL < 0.2
- Urine WBC < 10/hpf
- No infiltration on chest x-ray
- Fecal leukocytes < 5/hpf

Red Flags to be explained to parents at the time of discharge. The parents should be instructed to follow-up after 24-48 hours as per clinical condition in the primary health care system. A detailed account of danger signs should be explained to parents and if possible given a handout. It should be emphasized to them that if they notice or observe any of the following, they should come back to the ED immediately as it indicates worsening of the child's condition:

- Have breathing difficulty
- The lips, tongue or nails appear blue

- Crying continuously and inconsolable
- Refuse to eat or drink or appear too sick to eat or drink
- Vomiting whatever eating
- Has headache
- Has stiffness
- Develop skin rash
- Has severe abdominal pain
- Anything that worried parents from his baseline

**References and Further Reading,** click [here](#)

# Gastrointestinal Bleeding

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by Moira Carrol, Gurpreet Mudan, and Suzanne Bentley

## Case Presentation

*A 61-year-old man with a history of liver cirrhosis secondary to chronic EtOH abuse presents to the Emergency Department (ED) with a complaint of vomiting bright red blood that began prior to arrival. He arrives actively vomiting; a significant amount of blood is noted in his emesis basin. He is now complaining of dizziness and appears pale.*



Audio is available [here](#)

# Overview

Gastrointestinal bleeding (GIB) can be generalized into two categories based on the site of bleeding. Upper GIB (UGIB) is defined as any bleeding that occurs proximal to the ligament of Trietz near the terminal duodenum. Lower GIB (LGIB) is any bleeding that occurs distal to the ligament extending to the rectum. Most GIB seen in the ED is attributed to UGIB with an incidence of 90 per 100,000 population. LGIB, on the other hand, presents with a rate of 20 per 100,000 population. LGIB is more commonly seen in the elderly but has a wide range of presentations and causes. As a result, the approach to LGIB has been less standardized.

## Upper Gastrointestinal Bleeding (UGIB)

The most common causes of UGIB include peptic ulcer disease (PUD), erosive gastritis or esophagitis, esophageal or gastric variceal bleed and Mallory Weiss tears. Among these, PUD is the most common cause of UGIB presentations. Other less common causes include gastric malignancy, aortoenteric fistula, hematemesis and Dieulafoy's lesion, which is a large tortuous artery that can run very close to the gastric mucosa and can cause devastating bleeding.

## Lower Gastrointestinal Bleeding (LGIB)

LGIB is less common than UGIB in the ED. In fact, UGIB is identified in 11% of cases, whereas a lower GI source is found only in 9%. Diagnosis is sometimes elusive. LGIB can be the

result of diverticulitis, the most common cause of LGIB, or from hemorrhoids, colitis, anal fissures, inflammatory bowel disease including Crohn's disease and ulcerative colitis, colon cancer or angiodysplasia.

**Table 3.13** List of upper and lower G.I. bleeding causes

Table 1: List of Upper and Lower Gastrointestinal Bleeding Causes	
Upper G.I. Bleeding Causes	Lower G.I. Bleeding Causes
<ul style="list-style-type: none"> <li>• Peptic Ulcer Disease</li> <li>• Erosive Gastritis</li> <li>• Erosive Esophagitis</li> <li>• Esophageal or Gastric Varices</li> <li>• Mallory Weiss tears</li> <li>• Aortoenteric Fistula</li> <li>• Hematemesis</li> <li>• Dieulafoy's lesion</li> </ul>	<ul style="list-style-type: none"> <li>• Diverticular Disease</li> <li>• Colitis</li> <li>• Anal Fissures</li> <li>• Inflammatory Bowel Disease - Chron's and Ulcerative Colitis</li> <li>• Colon cancer</li> <li>• Angiodysplasia</li> <li>• Anorectal Fissures</li> </ul>

Please read G.I. bleeding chapter in Marx, John, Ron Walls, and Robert Hockberger. Rosen's Emergency Medicine-Concepts and Clinical Practice. Elsevier Health Sciences, 2013 for more detailed information about G.I. bleeding causes. Moira Carroll, Gurpreet Mudan, and Suzanne Bentley

## Critical Bedside Actions and Emergency Department Approach

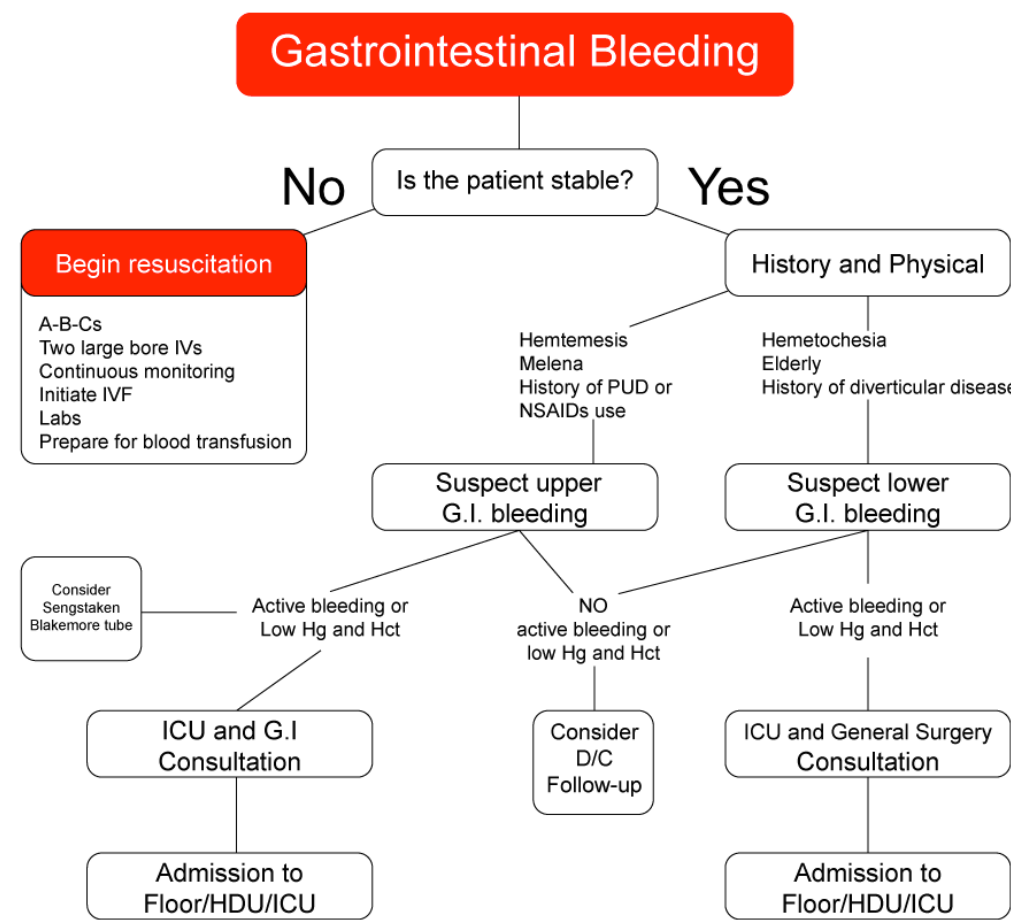
Just as the causes of acute GIB are diverse, so too are the possible presentations. As severe GIB can have a high risk of morbidity and mortality, patients with possible GIB should be identified quickly. In the ED, the patient should be evaluated for hemodynamic stability, as patients with brisk GI bleeding can rapidly decompensate. Assessment of patient stability includes evaluating the patient's general appearance, volume status, and vital signs. If deemed hemodynamically unstable upon initial clinical evaluation, begin resuscitation of the patient immediately. Please check for the general approach in Figure 3.5 (F1).



Figure 3.5 Approach to GIB

Figure 1: Approach to GI Bleeding

Moirá Carrol, Gurpreet Mudan, and Suzanne Bentley



First, do a rapid assessment and intervention of airway, breathing, and circulation (ABCs). Then, place two large bore IVs in preparation for IV crystalloid fluids infusion and possible blood product transfusion. Draw initial labs including a complete blood count, type and screen and type and crossmatch in case the patient will require blood product transfusion.

Indications for transfusion include hemodynamic instability despite crystalloid resuscitation, Hemoglobin (Hb) Hb < 9 g/dL in high-risk patients, Hb < 7 in low-risk patients. High-risk patients are considered those who are likely to rebleed or have severe hemorrhage, whereas low-risk patients are less likely. Various decision tools exist to help risk stratify patients based on multiple clinical criteria and lab values. Consider FFP to correct coagulopathy if present in a patient on anticoagulation or with severe liver dysfunction. Placing the patient on a cardiac monitor continually to assess changes in heart rate, blood pressure, and oxygen saturation is imperative.

### History and Physical Examination Hints

After initial stabilization, the next step is to determine the probable cause of the bleeding in order to treat appropriately and disposition the patient. The history and physical can guide you towards identifying the likely source of the bleeding and direct the necessary treatments and consultations. UGIB can often be definitively managed by gastroenterologists. Lower GI bleeding, however, might require interventions by general surgeons, gastroenterologists or interventional radiologists.

Certain complaints are unique to GIB. One of the most important pieces of history is to assess complaints of active bleeding. Hematemesis is virtually diagnostic of UGIB. Hematemesis is defined as bloody vomit, either appearing as bright red blood or as coffee ground emesis. Melena, or dark, tarry stool, is also a

sign of UGIB. In patients with UGIB, between 90-98% presented with either melena or hematemesis. Alternatively, hematochezia is defined as blood within or around the stool. However, hematochezia can sometimes be the result of a brisk UGIB. Diagnosis can also be confounded if there is slow peristalsis in the setting of an LGIB.

Therefore, start by assessing the context of the bleed as it can give you clues to its origin. For example, patients who have a bleed secondary to PUD might have a history of an ulcer, might complain of acid reflux or have a recent history of frequent NSAID use. Patients with gastric or esophageal varices might describe a history of or risk factors for liver disease, such as daily alcohol abuse, or have other pathognomonic signs of portal hypertension. A history of intractable vomiting in the setting of hematemesis may suggest Mallory Weiss tears as the cause.

Similarly, a patient who complains of blood in the stool with a history of constipation suggests bleeding caused by the diverticular disease. Recent diarrheal illness can be found in infectious colitis. An elderly patient presenting with weight loss or anorexia is concerning for malignancy. The duration and timing of the bleeding are important to determine. Brisk or continued bleeding can alert to the need for resuscitation or emergency intervention. Finally, the provider must characterize and quantify the bleeding. In a complaint with multiple pathologic causes, a good history and physical exam are paramount.

## Emergency Diagnostic Tests and Interpretation

### Laboratory Studies

The most important lab tests for risk stratification for patients with acute GIB are the hemoglobin (Hb) and hematocrit (Hct), coagulation studies, and BUN to Creatinine ratio. A type&screen is recommended as well in case of

expected blood transfusion. Initially, Hb and Hct may be within normal limits. The values might not immediately reflect blood loss after an acute hemorrhage and, therefore, should be repeated. Higher mortality and incidences of rebleeding were found in patients with Hb < 10 g/dL. Additionally, many recommend using the Hb and Hct to inform the decision to type and crossmatch blood versus only drawing a type and screen.

In a patient without kidney disease, a BUN to Creatinine ratio that is elevated to greater than or equal to 36 is strongly associated with UGIB. As blood is digested, the BUN is reabsorbed into the circulation leading to elevated serum levels. Below 36, however, the ratio has no positive or negative predictive value. BUN/Cr >36 can be helpful in the diagnosis of an occult UGIB in those patients who present without classic signs of GI bleeding.

The role of nasogastric (NG) lavage and aspiration in the diagnosis of GI bleeding

has been controversial. NG aspiration positive for blood is highly predictive of a UGIB. However, it has not proven to be sensitive. Placement of an NG is not a benign procedure as there are risks including perforation and discomfort.

### Fecal occult blood test

Performing a fecal occult blood test via a rectal exam is important in the setting of a GIB. This bedside test can confirm whether or not blood is present in the stool, confirming the presence of a GIB. Unfortunately, it is not specific to UGIB or LGIB; however, often the presence of melena or bright red blood can help guide diagnosis.

### Upper Endoscopy

Upper endoscopy is overwhelmingly diagnostic and usually therapeutic for UGIB. Consultation with gastroenterology is necessary for the emergent scope of patients with continued bleeding and suspected UGIB. These specialists can immediately diagnose and treat the source of bleeding. Stable patients with

suspected UGIB can undergo endoscopy as an inpatient. Early endoscopy, within the first 24 hours of presentation, is associated with shorter hospital stays and early instigation of appropriate treatment. However, most UGIB resolves without this intervention. **Figure** shows a duodenal ulcer (Deep demarcated ulceration with a visible vessel on base (Forrest Iia) Source: Lai, WEO Endoscopy Atlas, Date: 2012-12-25.

### Colonoscopy

Colonoscopy can be helpful in the diagnosis of LGIB and is an effective first-line test, but it is not a gold standard in the diagnosis of LGIB. A diagnosis is made by colonoscopy in 75% of cases. Typically, lower GI scopes are not performed emergently but can be performed later during hospitalization or as an outpatient. **Figure** shows colonoscopy, bleeding from multiple diverticular outpouchings. Source: American Family Physicians, Wilkins et al. Diverticular bleeding (please see **Figure** in their manuscript).

### Tagged Red Blood Cell Scan

Tagged Red Blood Cell Scan is a second line study that can assist in the diagnosis of more indolent and continued bleeding. Scanning within the recommended two-hour window after the injection has high rates of positive diagnosis in 95-100% of cases but after the recommended time period the test is significantly less effective.

### Medications

Only a few medications have been shown to be influential in the acute management of GIB. Pantoprazole is indicated for a UGIB in the setting of PUD. It is given as an 80 mg bolus followed by an infusion at a rate of 8 mg/hour. If variceal bleeding is known or suspected, consider starting Octreotide or other somatostatin analog. Octreotide is given as a 25-50 mcg bolus, then 25-30 mcg/hr infusion. In patients with cirrhosis, antibiotics such as Ceftriaxone, Amoxicillin-clavulanate or Quinolone should be given.

## Procedures

Sengstaken-Blakemore Tube is a device that is inflated in the esophagus to tamponade uncontrolled bleeding caused by varices. It is used as a measure of last resort because of the high complication rate. EM CRIT – [VIDEO](#) – Blakemore Tube Placement for Massive Upper GI Hemorrhage.

## Disposition of Patient with Gastrointestinal Bleeding

Finally, the patient with GI bleeding will need to have a disposition based on the resuscitation and findings of the workup. Unstable patients or those with active GIB and rapidly decreasing Hb and Hct levels on reassessment should receive a consult from the intensive care unit. If a patient and Hb/Hct remain stable, admission to a regular medical floor or possible discharge home with close, appropriate follow-up may be considered. Appropriate follow-up should be timely with a gastroenterologist. Prior to discharge, patients should be encouraged to avoid medications and

behaviors that may increase the risk of bleeding again, such as NSAIDs and alcohol.

**References and Further Reading**, click [here](#)

# Headache

by Matevz Privsek and Gregor Prosen

## Case Presentation

*A 52-year old male comes to the ED with a severe headache. A triage nurse gives you his chart and says that his vital signs are normal, but he does not look well. You start to question the patient, and the following history is obtained: his headache started approximately six hours ago. He was working in his office when he started to feel squeezing-like sensation in his head. The pain has gotten worse since then, but it is still tolerable. It is independent of any physical activity or position. He already had a few similar episodes of this kind of headache in the past two years, but now the pain does not go away after aspirin as it did previously. He denies trauma as well as any associated symptoms, e.g. no visual disturbances, hearing loss, weakness, dizziness, stiff neck, loss of consciousness. He is otherwise a healthy, non-smoker, with no regular therapy or known allergies. His clinical exam is*



Audio is available [here](#)



unremarkable. Conscious, GCS 15, alert and oriented, normal skin color. Blood pressure 135/82 mm Hg, pulse 78/min, 14 breaths/min, SpO2 99%, body temperature 36,4 °C. Neurologic exam shows no declines from normal, as well as the rest of the physical exam.

You set up an intravenous cannula, draw blood for testing, and gave the patient some parenteral analgesics (metamizole 2.5 g, ketoprofen 100 mg) along with 500 ml of normal saline. You put him into the observation room. Lab results (complete blood count, basic biochemistry panel) came

back in 2 hours and are completely normal. The patient now feels much better, with almost no headache at all. Repeated vital signs and clinical exam are again unremarkable. You explain to the patient that most likely he had a tension headache, warn him about red flags regarding headaches, and discharge him home with a prescription for peroral analgesics with a follow-up at his general physician.

## Introduction

Headache is a subjective feeling of pain, crushing, squeezing or stabbing anywhere in the head. They are typically divided to primary and secondary headaches. The most important task emergency physicians have is to exclude any potentially lethal causes of headache.

According to some data, around 85% of the adult population experience headaches at least occasionally, and 15% does so regularly. A headache is the chief complaint in around 3-5 % of all emergency departments (ED) visits.

## Pathophysiology

The pain in the head originates either from the meninges, blood vessels or surrounding tissues; the brain parenchyma itself is insensitive to pain. Because most of the pain is mediated through the fifth cranial nerve, the patient's ability to localize the pain is often poor. More specific localization of the pain is associated with specific

inflammation in a specific structure (e.g. sinusitis).

## Etiology

A vast number of diseases can cause a headache. We divide them into primary headaches, in which the headache is the disorder in itself, and secondary headaches, in which headaches are caused by various exogenous disorders (Table 3.14).

**Table 3.14** Etiology of headaches

iEM Table 1: Etiology of headaches	
Primary headaches	Secondary headaches
<ul style="list-style-type: none"> <li>• Tension headache</li> <li>• Migraine headache</li> <li>• Cluster headache</li> </ul>	<p><b>Vascular causes</b></p> <ul style="list-style-type: none"> <li>• Subarachnoid hemorrhage (SAH)</li> <li>• Intracerebral hemorrhage (ICH)</li> <li>• Subdural / epidural hematoma (SDH, EDH)</li> <li>• Stroke</li> <li>• Brain venous thrombosis</li> <li>• Arterial-venous malformations</li> <li>• Temporal arteritis</li> <li>• Dissection of carotid / vertebral artery</li> </ul> <p><b>Infections</b></p> <ul style="list-style-type: none"> <li>• Meningitis, encephalitis, brain abscess</li> <li>• Systemic infection</li> <li>• Sinusitis, dental and ear infections</li> </ul> <p><b>Drugs and toxins</b></p> <ul style="list-style-type: none"> <li>• Intoxication with carbon monoxide</li> <li>• Misuse of analgesics</li> <li>• Nitrates and nitrites</li> <li>• MAO inhibitors</li> </ul> <p><b>Brain tumor</b></p> <ul style="list-style-type: none"> <li>• Benign intracranial hypertension</li> </ul> <p><b>Ophthalmologic conditions</b></p> <ul style="list-style-type: none"> <li>• Glaucoma (acute, angle-closed)</li> <li>• Iritis</li> <li>• Optic neuritis</li> </ul> <p><b>Endocrine and metabolic conditions</b></p> <ul style="list-style-type: none"> <li>• Pheochromocytoma</li> <li>• Hypoxia</li> <li>• Hypercapnia</li> <li>• Hypoglycemia</li> </ul> <p><b>Muscular causes</b></p> <ul style="list-style-type: none"> <li>• Post lumbar puncture</li> <li>• Hypertension</li> </ul> <p><b>Obstetric</b></p> <ul style="list-style-type: none"> <li>• Preeclampsia</li> </ul>

Adopted from Ropič P, Glavobol. In Prosen G, Baznik Š, Mekš D, Strnad M, editors. Šola urgence, zbornik 1. letnika. Ljubljana: Slovensko združenje za urgentno medicino; 2014. p. 117-21., by Matevž Privsek and Gregor Prosen

Around 50% of patients with a headache in the ED have a tension-type headache, 10% have a migraine-type headache, 8% have a secondary headache, and 30% of headaches remain

unknown. It is estimated that less than 1% of patients with headache have a serious, life-threatening underlying disease.

## Management

### Critical Bedside Actions And General Approach

Regardless of the patients' chief complaint, an emergency physician's first task is assessing a patient's condition and vital signs, and stabilize him/her, if necessary. After the patient is stabilized, we continue with establishing the chief complaint, precise history, and physical exam, setting the working diagnosis and list of differential diagnoses, and diagnostic and treatment plan.

### Differential Diagnosis

An emergency physician has to exclude life-threatening causes of headache, based on history, physical exam, and diagnostic tests. Subarachnoid hemorrhage (SAH), meningitis, encephalitis, carbon monoxide poisoning, and temporal arteritis considered as critical diagnoses. Other emergency causes of headache are shunt failure, sub or epidural hemorrhage, tumor/mass lesions, mountain sickness, glaucoma, sinusitis, brain abscess, anoxic headache, anemia, and hypertensive crisis.

# History And Physical Examination Hints

The leading symptom has to be thoroughly “dissected” and a focused medical history must be obtained. Useful mnemonics for this are “SOCRATES” and “SAMPLE” (Table 3.15).

**Table 3.15** Getting fast and effective history about chief complaint

Table 2: Mnemonic “SOCRATES” and “SAMPLE” for dissecting chief complaint		
<b>S</b>	site	site of pain/symptom (frontal, temporal, occipital, hemicranial)
<b>O</b>	onset	how did the symptom began (suddenly, slowly)
<b>C</b>	character	nature of the pain (sharp, stabbing, squeezing, crushing)
<b>R</b>	radiation	does the pain radiate somewhere or it remains where it started?
<b>A</b>	associations	nausea, vomiting, sensitivity to light or voices, stiff neck, fever ...
<b>T</b>	time course	how did the symptom evolve since it began (increasing in severity, waxing and waning)
<b>E</b>	exacerbation & alleviating factors	are there any factors that make the pain easier or worse (physical activity, rest, position, noise, light)
<b>S</b>	severity	how severe was the pain when it began, and how it is now (VAS)
<b>S</b>	signs & symptoms	chief complaint and its associations
<b>A</b>	allergies	known allergies to medications
<b>M</b>	medications	prescribed and OTC medications, drugs
<b>P</b>	past illnesses	relevant past medical and surgical history
<b>L</b>	last oral intake	important especially if surgical procedure is considered
<b>E</b>	events leading up to presenting illness	how it all began?

VAS: Visual analogue scale, produced by Matevz Privsek and Gregor Prosen

When taking the history of presenting illness, one must be especially focused on:

- marked variation in headache pattern (if already had previous episodes of headache),
- sudden onset of “thunder-clap” or “lightning strike” headache (possible SAH),

- patient’s activity at the onset of the pain (headaches associated with exertion are suggestive of vascular bleeding),
- history or possibility of head trauma suggests possible epidural or subdural hematoma, traumatic SAH, skull fracture, and closed-head injury (e.g. diffuse axonal injury),
- in immunocompromised or HIV-infected patients one must consider for brain abscess, toxoplasmosis, or cryptococcal meningitis,
- the severity of headache is not useful to accurately distinguish differential diagnoses; it is more helpful in monitoring the patients’ response to treatment. However, relieving headache with pain medications will not eliminate severe, secondary causes.
- the character of the pain alone is not enough to adequately differentiate one type of headache from another,
- location of headache is only helpful when the patient identifies a specific, well-localized area,
- nausea and vomiting are completely nonspecific and points towards the intensity of the complaint.

**Tension headache** is the most common. Usually, it is bilateral, non-pulsating, and mild to moderate severity, not related to

physical activity. Pain is blunt, squeezing-like. It is most common in the afternoon or evening.

**Migraine** starts gradually and can last up to three days. Pain is unilateral, pulsating, and severe, often worse with physical activity. Vomiting, as well as photo and noise sensitivity, often accompany it. Aura (transitional vision disturbances, paresthesias, speech disturbances) can be present before an attack, but it is rare. It is more common in females.

**Cluster headache** is extremely rare (overall prevalence around 0,4%). Its characteristics are extremely severe, unilateral pain, limited to orbital, supraorbital or temporal regions. It is of short duration (few minutes up to two hours), during which the patient cannot be still.

### Emergency Diagnostic Tests And Interpretation

The majority of patients with headache do not require any additional testing. The emergency physician has the following options: blood tests, **head CT**, **lumbar puncture** and **cerebrospinal fluid analysis** (Table 3.16).

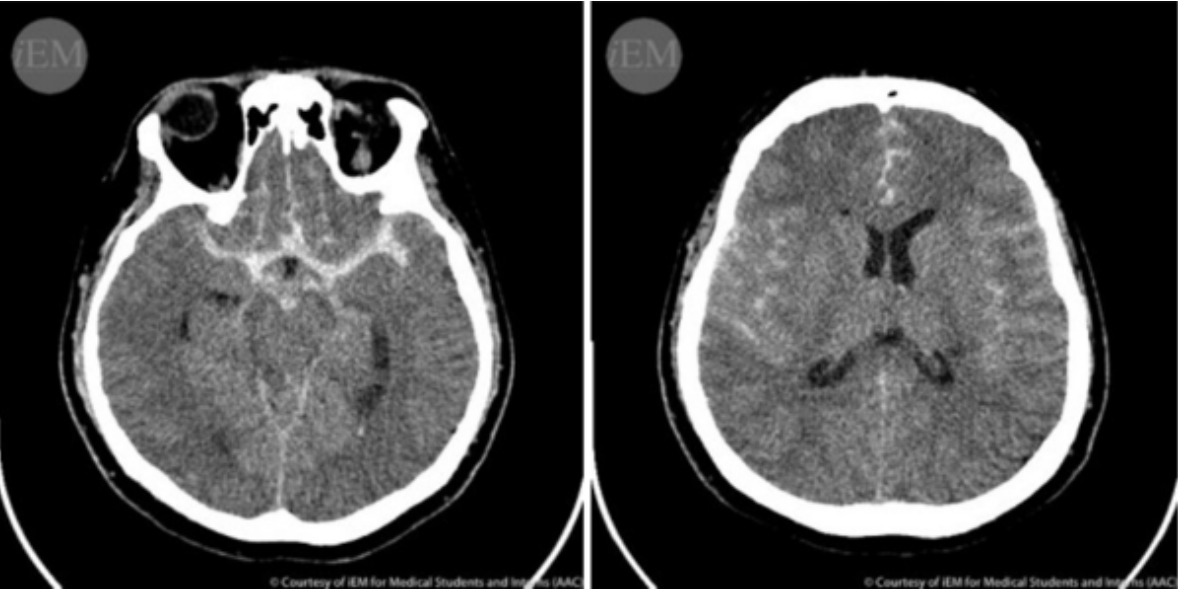
It is important to know that head CT scan misses 6-8 % of patients with SAH, its' sensitivity for detecting SAH is reduced by 10% if the symptoms began over 12 hours ago, and almost by 20-30% if they began 3-5 days ago.

**Table 3.16** Emergency diagnostic tests for headache

iEM Table 3: Emergency diagnostic tests for headache		
Diagnostic test	Results	Interpretation*
Blood tests	Anemia	Hypoxia
	Leukocytosis	Infection
	C-reactive protein	Infection (viral vs. Bacterial)
	Erythrocyte sedimentation rate	Temporal arteritis
Head CT	Increased size of ventricles	Increased CSF pressure
	Blood in subarachnoid space	Subarachnoid hemorrhage
	Blood in epi- or subdural space	Epi- or subdural hematoma
	Bleeding into brain parenchyma	Intracerebral hemorrhage
	Mass lesion	Secondary traction headache
Lumbar puncture and csf analysis	Increased csf pressure	Mass lesions, shunt failure
	Increased proteins	Tumor / structural lesion
	Increased red blood cells	SAH
	Increased white blood cells	Infection
	Positive gram's stain	Infection
	Decreased glucose	Infection

\* interpretation has to be made in accordance with clinical picture. CT: computerized tomography; CSF: cerebrospinal fluid; SAH: subarachnoid hemorrhage. Adopted from Russi (2010), Ropić P, Glavobol (2014), please see references for more information. Matevz Pihovek and Gregor Prosen

**Image 3.16** SAH





On the other hand, head CT scan within 6 hours of onset of the pain has almost 100% sensitivity for SAH; therefore, lumbar puncture is reasonable only when head CT does not confirm the diagnosis, yet the clinical picture is highly suspicious for SAH.

## Emergency Treatment Options

Pain relief is the first and most important thing of management, at least initially in an undifferentiated headache. Depending on the severity and associated symptoms (e.g. vomiting) we can choose between paracetamol and non-steroid anti-inflammatory drugs, metamizole, and opiates. Analgesics can be given per oral or parenterally (Table 3.17). For specific headache treatment options, see Table 3.18.

**Table 3.17** Analgesics for headache

iEM Table 4: Some analgesic options for headache			
Drug	Adult dosing	Pediatric dosing	Special considerations
Paracetamol (acetaminophen)	500-1000 mg PO QID	6-12 years: 250-500 mg PO QID  < 6 years: use syrup or rectal suppositories	Hepatotoxic
Diclofenac	75 mg PO BID or 75 mg IM BID	not recommended under 14 years of age	GIT disturbances
Naproxen	550 mg PO BID or TID, if tolerated	not recommended under 16 years of age	GIT disturbances
Ketoprofen	100 mg in 100 mL of NS IV, BID	not recommended under 15 years of age	GIT disturbances
Metamizole	2,5 g in 100 mL of NS IV, BID or 500-1000 mg PO QID	can be used beyond 3 months of age, see weight specific dosages	Agranulocytosis, hypotensive reaction
Tramadol	50-100 mg IV QID or 100-200 mg PO BID	1-2 mg/kg IV QID	
Morphine	2-15 mg IV QID or 10-60 mg PO QID	> 12 years: 0,05-0,1 mg/kg IV QID or 10-20 mg PO QID	
Ketamine (s-ketamin)	0.2 mg/kg IV (0.1 mg/kg IV)	same	for acute relief (usually 5-10 mg IV enough)

QID, four times daily; PO, per os; IM, intramuscular; BID, two times daily; GIT, gastrointestinal; TID, three times daily; NS, normal saline; IV, intravenous.

Adopted from European Medicines Agency. European Medicines Agency (2016), please see references for more information. Matevz Privsek and Gregor Prosen

**Table 3.18** Initial treatment options for primary headaches

iEM Table 5: Initial treatment options for primary headaches	
Cause of headache	Treatment
Tension headache	<ul style="list-style-type: none"> <li>Paracetamol or NSAIDs of choice</li> <li>If severe or persist add more potent analgesics or treat as migraine</li> </ul>
Migraine	<ul style="list-style-type: none"> <li>Dihydroergotamine 2,5 mg PO BID/TID for a few weeks or</li> <li>Sumatriptan 50-100 mg PO once (if effective but the pain still persist, can repeat in 24 hours; if not effective, discontinue the drug)</li> <li>***Do not combine dihydroergotamine with triptans!</li> <li>Paracetamol, nsais of choice, potent analgesics</li> <li>If nausea and/or vomiting, add metoclopramide (10 mg IV)</li> <li>If symptoms persist, consider adding magnesium sulfate (1-2 gr IV)</li> <li>Based on local policy, add dexamethasone 10 mg IV for recurrence prevention</li> </ul>
Cluster headache	<ul style="list-style-type: none"> <li>High flow O<sub>2</sub> (&gt; 10 L/min)</li> <li>Dihydroergotamine or sumatriptan</li> <li>Nsais can be useful for prevention</li> </ul>

Adopted from Rapić P, Glevobol (2014) and European Medicines Agency. European Medicines Agency (2016), please see references for more information. Matevz Privsek and Gregor Prosen

Treatment for secondary headaches is directed towards the cause (e.g. surgery, antibiotics, antihypertensives), but we must not forget about pain relief!

## Pediatric, Geriatric, Pregnant patient, and Other Considerations

All the principles for adults can be safely applied to pediatric, geriatric or pregnant patients with headache, with regards to differential diagnosis (e.g. geriatric patient has increased likelihood for a vascular headache). Venous sinus thrombosis should be kept in mind for pregnant and peripartum patients. Sinusitis is one of the common cause of childhood headaches.

## Disposition Decisions

The vast majority of patients with headache can be discharged home with a prescription for analgesics and a close follow-up. These patients are those in whom ED therapy was successful in



pain relief, have normal clinical exam and vital signs, and no serious illness has been identified or suspected. All other patients require additional work-up or admission.

**References and Further Reading**, click [here](#)

# Multiple Trauma

---

by Pia Jerot and Gregor Prosen

## Case Presentation

*A 28-year old male was a restrained driver in a head-on motor vehicle collision. He was entrapped and extricated from the vehicle. Transient loss of consciousness was reported. He complains of severe chest pain, abdominal pain, and right upper leg pain.*



Audio is available [here](#)

## Critical Bedside Actions and General Approach

Multiple trauma patients are primarily stabilized by the ambulance crew on the field according to Prehospital Trauma Life Support (PHTLS) or International Trauma Life Support (ITLS) algorithm. This chapter is about the approach to multiple trauma patients in the resuscitation room and mainly focuses primary survey.

### Preparing For Patient Arrival

The ambulance service has to provide information to the trauma center for the arrival of the seriously injured patient. The trauma team puts on protective clothing (rubber latex gloves, plastic aprons, eye protection, etc.). A team leader should brief the team and make sure that every member knows his role and all necessary equipment is ready.

### Primary Survey

In the first few minutes, a primary survey has to be done. The primary survey is a structured assessment in which we identify and immediately treat conditions that are life-threatening. The primary survey should always be the same, following the ABCDE algorithm listed below.

**A: airway and cervical spine control**

**B: breathing**

**C: circulation**

**D: disability**

**E: exposure**

In case of massive external hemorrhage, “C” takes advantage over A and B. When the bleeding is controlled, we can continue with A and B.

### A – Airway and cervical spine control

When the patient arrives at the trauma center, talk to the patient and quickly assess his consciousness and airway. If the patient is conscious and talking with a normal voice, his airway is adequate for a period. If it is obstructed, the airway has to be secured. While securing the airway, C-spine has to be protected, especially when we are dealing with a patient with neck pain, focal neurological signs, coma, suspected head injury or a history of high-speed impact.

Do not delay C-spine and vertebra stabilization in a trauma patient with proper size of c-collar, side pads, and trauma board..

The airway can be temporarily opened with a jaw thrust or basic adjuncts such as nasopharyngeal or oropharyngeal airway. If the patient’s airway is still compromised, it should be secured by endotracheal intubation or surgical airway. Indications for endotracheal intubation are listed in Table 3.19. Intubated patients should be monitored by continuous capnography.

**Table 3.19** Indications for ET intubation

**iEM** **Table 1: Indications of Endotracheal Intubation**

- A: airway obstruction, airway protection
- B: inadequate ventilation, hypoxemia, hypercapnia
- C: persistent hypotension, shock
- D: severe head injury (GCS 8 or less)
- E: uncooperative patient
- F (fire): thermal injury to the upper airway

Adopted from Driscoll & Skinner (2013); Betz et al. (2011), please see references for more information, Pia Jerot and Gregor Prosen

### B – Breathing

All trauma patients should be given 15 L O2 via non-rebreather mask. The respiratory rate has to be evaluated. Expose the chest, and inspect for any deformities, wounds, bruising, asymmetrical movement or flail chest. The chest has to be palpated for any crepitus or subcutaneous emphysema. The lungs should be auscultated to assess the presence of breathing sounds bilaterally.

Chest injuries that can impair breathing have to be identified and treated. Life-threatening thoracic conditions are tension pneumothorax, massive haemothorax, cardiac tamponade, open chest wound, **flail chest** (video). If any of those conditions are found, immediate action is needed.

We can also use ultrasound at this moment to identify **pneumothorax**, **haemothorax**, and **cardiac tamponade**.

### C – Circulation and hemorrhage control

The main objective of this step is to identifying shock situation in the patients. If there is massive external hemorrhage, it should be controlled before we proceed with A and B assessment. Radial pulses, heart rate and blood pressure (BP) should be assessed at that stage. The patient should be attached to the monitor. If we are dealing with a major trauma patient, at least two large bore (14G or 16 G) IVs have to be established. As an alternative, intraosseous line can be used.

“Permissive hypotension” with systolic BP 80-90 mmHg should be maintained when bleeding is not controlled (internal bleeding). If the patient is hemodynamically unstable, fluid resuscitation should begin with 1 L of warm isotonic fluid, either normal saline or lactated Ringer’s. At this moment, it is better to keep in your mind that normal vitals are considered stage I shock which patients could lose up to 750 cc blood. So, starting fluid support to patients suspected multiple trauma is a standard approach regardless of their vital signs. If the patient is still unstable after 1 Liter of fluid or is having ongoing blood loss, we should administer a transfusion of O-negative blood and order type-specific blood. The patients who have an identified bleeding source will usually require surgical intervention. Do not delay surgery departments’ involvement. Internal bleeding in the thorax, abdomen, pelvis or around fractures of long bones (particularly femur) should also be evaluated. For identifying bleeding into abdominal, pleural, or pericardial cavities, we can use the bedside

ultrasound (**e-FAST exam**). In the hemodynamically unstable patient with normal e-FAST, one should think pelvic injuries.

**Image 3.17** Positive eFAST exam (peri-splenic free fluid)



### D – Disability

In the primary survey pupil size, symmetry and reactivity should be assessed. To detect the level of consciousness, the patient has to be assessed by AVPU scale. It stands for A: Awake, V: responds to voice or verbal commands, P: responds to painful stimuli, U: unresponsive.

If there is time, Glasgow Coma Scale (GCS) can be used to assess the level of consciousness. Otherwise, GCS is assessed in the secondary survey. GCS is listed in Table 3.20.

**Table 3.20** Glasgow Coma Scale

iEM Table 2: Glasgow Coma Scale						
Score	1	2	3	4	5	6
Eye	Does not open eyes	Opens eyes in response to painful stimuli <a href="https://en.wikipedia.org/wiki/Pain_stimulus">https://en.wikipedia.org/wiki/Pain_stimulus</a>	Opens eyes in response to voice	Opens eyes spontaneously		
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	
Motor	Makes no movements	Extension to painful stimuli	Abnormal flexion to painful stimuli	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

Adapted from Betz et al. (2011), please see references for more information, Pia Jerot and Gregor Prosen

And finally, patients should be evaluated for any lateralized motor deficits which motor part of the GCS may help for this. An important clue, the patients can not move their extremities because of focal injuries.

In this stage, blood glucose and body temperature should also be checked.

### E – Exposure

All clothes have to be removed from the patient so that hidden injuries and bleeding can be identified. The patient has to be log-rolled. In a log-roll, back of the head, neck, posterior chest, lower back should be inspected, palpated and auscultated as appropriate. If necessary, the rectal examination can be applied at this moment. We have to avoid hypothermia during this stage.



# Secondary Survey

The secondary survey can be done after the primary survey and when the patient is responding to resuscitation. It consists of taking history (see “SAMPLE” mnemonic below), head to toe assessment (including log-roll), interpreting results of investigations, formulating a management plan for the patient and documenting all findings.

S: symptoms – social

A: allergies

M: medications

P: past medical history

L: last meal

E: environment and events

# Differential Diagnoses

There are critical conditions which should have been diagnosed during the primary survey. These are;

- A: Airway obstruction caused by c-spine injury, foreign body, etc.
- B: Tension pneumothorax, flail chest, massive haemothorax, chest wound

- C: Shock because of cardiac tamponade, intra-abdominal, intrathoracic, intrapelvic hemorrhage, external hemorrhage
- D: Head injury
- E: Fractures, dislocations and small wounds

# History and Physical Examination Hints

History and physical examination hints to help “rule in or rule out” differential diagnoses are listed in Tables 3.21.

**Table 3.21** Lorem Ipsum dolor amet, consectetur

Table 3: History and Physical Examination Hints	
Primary Survey Step	Comments on History and Physical Examination
<b>Airway</b>	
Obstructed airway	Patient is not awake. Patient is not speaking normally. Dyspnea. Abnormal breathing sounds (wheezing, stridor). Facial or neck trauma. Head injury
C-spine injury	Neck pain Focal neurological signs History of high speed impact Head injury
<b>Breathing</b>	
Tension pneumothorax	Hypotension Severe dyspnea Decreased breathing sounds Subcutaneous emphysema Deviated trachea
Flail chest	History of high speed impact Paradoxical or reverse motion of a chest wall segment
Massive haemothorax	Decreased breathing sounds Signs of shock
<b>Circulation</b>	
External hemorrhage	Visible blood Signs of shock
Internal bleeding	Fractured ribs, unstable pelvis Bruises Distended abdomen Signs of shock
Cardiac tamponade	History of high speed collision Bruises on chest Hypotension Distended neck veins Muffled heart sounds

As an example, any patients with head, neck, facial injury or having abnormal breath sounds should be suspected of an

obstructed airway. Any patients with severe shortness of breath, decreased or absent breath sounds on the lung, and hypotension is a tension pneumothorax until proven otherwise.

## Emergency Diagnostic Tests and Interpretation

### Laboratory tests

Laboratory evaluation of the trauma patient is used for assessing the adequacy of resuscitation, for determining the proper transfusion products and the onset of coagulopathy and for baseline values for follow-up studies.

When IV access has been established, 20 mL of blood should be taken for a full blood count, urea, electrolytes and for blood group type or full cross-match. An **arterial blood** should also be taken for blood gas and pH analysis, but it can be taken at the end of the primary survey. If there is a very limited amount of blood taken, blood group type and cross-match should be the only test for the patient.

### Imaging

The **e-FAST (Extended Focused Assessment with Sonography for Trauma)** should ideally be a part of the primary survey, especially for unstable patients. It can be used for evaluating pneumothorax, free fluid in thorax and abdomen and to identify cardiac tamponade.

A trauma patient should be radiographically evaluated with chest and pelvic radiographs. **C-spine X-ray** was traditionally used. Nowadays, any low-risk criteria violation is considered for computerised tomographic evaluation for the cervical spine. Cervical spine imaging can be delayed if there are no neurologic findings or persistent moderate hypotension. For assessing C-spine and other injuries, **CT scan** is the best choice in the acute setting, when the patient is stable.

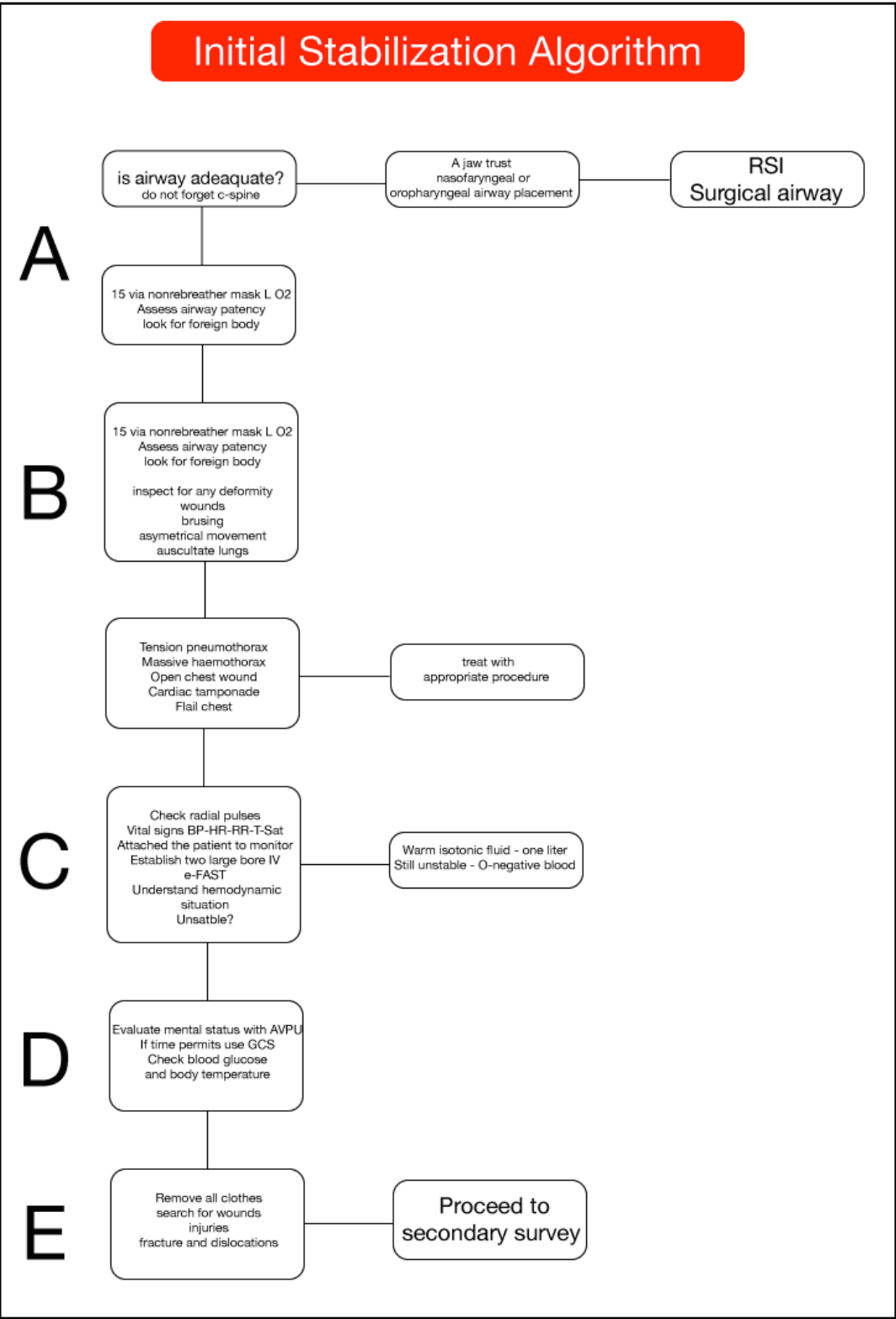
Imaging studies of the thoracolumbar spine and extremities can be delayed until higher priority assessments and interventions are complete.

## Emergency Treatment Options

### Initial stabilization

Initial stabilization should be done during primary survey. The algorithm is shown in Diagram 3.2. The goal is every abnormality detected during the primary survey should be fixed.

Diagram 3.2 Initial stabilization



Produced from Driscoll&Skinner (2013), Betz et al. (2011) and Bhangu (2010), please see references for more information, by Pia Jerot and Gregor Prosen

Medications

Drugs needed for RSI

For **RSI**, you will need a pretreatment agent (mainly fentanyl), induction agent (mainly ketamine or etomidate) and paralytic agent (mainly rocuronium). Dosages and characteristics of drugs are listed in Table 3.22. Doses must be adjusted in the hypotensive or shocked patient.

Analgesia

For **analgesia**, opioids or ketamine can be used. Dosages and characteristics of drugs are listed in Table 3.22.

Table 3.22 Drugs for RSI and analgesia

iEM Table 4: Drugs Needed For RSI and Analgesia						
Drug	Adult dose	Pediatric dose	Onset	Duration	Pros	Cons
RSI						
Pretreatment						
Fentanyl	0.5-3 mcg/kg IV	0.3-5 mcg/kg IV	Immediate 1-5 min	-	Neuroprotection in head injury	May cause hypotension.
Induction						
Ketamine	1-2 mg/kg IV	1-2 mg/kg IV	60-90 sec	10-20 min	Provides analgesia, bronchodilatory effects, helps to maintain BP in hemodynamically unstable patient.	
Etomidate	0.3 mg/kg IV	0.1-0.3 mg/kg	0.5-1min	3-5 min	Does not alter hemodynamics or ICP	Does not provide analgesia
Paralytics						
Rocuronium	1.2 mg/kg	0.6 mcg/kg IV	60 s	30-45 min	Can be reversed by sugammadex, minimal effect on hemodynamics.	Allergic reaction.
Succinylcholine	1.5 mg/kg	1-2 mcg/kg IV	45-60 s	4-5 min	Ideal if need to asses patients neurology in an intubated patient (duration 6-10 min)	Numerous contra-indications, bradykardia, hyperkalemia
Analgesics						
Morphine	0.01-0.2 mg/kg IV	0.1-0.2 mg/kg IV	1-5 min	20-180 min	Antidote: naloxone	Can cause nausea and vomiting.
Fentanyl	0.5-3mcg/kg IV	0.3-5 mcg/kg IV	Immediate 1-5 min	15-20 min	Neuroprotection in head injury	May cause hypotension

IV, intravenous. Produced from Nickson C (2015), Lafferty KA (2016) and Kupnik D (2008), please see references for more information, Pia Jerot and Gregor Prosen

## Procedures

All procedures for initial stabilization should be done in the primary survey.

**Airway:** If the patient's airway is compromised after a jaw thrust, nasopharyngeal or oropharyngeal tube placement, then orotracheal intubation is indicated. **Rapid sequence intubation** should be done. If it is unsuccessful, the surgical airway should be established.

**Breathing:** If tension pneumothorax is found in the primary survey, it should be decompressed immediately. Tube thoracostomy should be followed this procedure. If massive haemothorax is found, thoracostomy should be performed. However, in this situation, please make sure that the patient was supplied with enough volume and blood. Informing trauma surgery or thoracic surgery for potential thoracotomy risk is a wise approach.

**Circulation:** If massive bleeding is found, it should be controlled immediately. If cardiac tamponade is found, it should be treated with **pericardiocentesis**.

## Pediatric, Geriatric, and Pregnant Patient Considerations

### Pediatric Patient Considerations

**Airway:** Airway should be checked the same as in an adult patient. When positioned flat on a stretcher, the occiput can flex

the neck and the floppy upper airway which can result in occlusion of the airway. To prevent this, place a pad under the torso of children younger than eight years.

**Breathing:** Normal respiratory rates vary in different ages. Newborn respirates 25-50 breath per minute while 6 months to 12 year kids respire 15-30 breath per minute.

**Circulation:** Brachial and femoral pulses are usually easy to feel. A weak, rapid pulse with a rate over 130 is a sign of shock in children all ages except neonates. Children have a strong compensatory mechanism in early shock and later deteriorate very quickly. When giving fluid resuscitation, give 20 mL/kg in each bolus.

**Disability:** When evaluating an injured child, the care provider should remember that children of various ages have different cognitive skills and interact differently. For assessing child's neurological status, special GCS is used.

### Geriatric Patient Considerations

The geriatric patient should be assessed and treated by ATLS protocol for adults. Because of multiple comorbidities in this age group, the patients may need special considerations such as medications which affect vital signs and basal laboratory results which affect the decision on imaging modalities (high kidney functions). However, life-threatening situations have priority, and immediate actions should be taken as with normal adults.

## Pregnant Patient Considerations

Primary survey is the same for the pregnant patient as for other patients.

The normal heart rate of a pregnant patient is 10-15 beats faster than usual, and the blood pressure is 10-15 mmHg lower; so, normal vital signs can be mistaken for a shock. A blood loss of 30-35% can occur before there is a significant fall in blood pressure.

## Disposition Decisions

### Admission criteria

The majority of patients will be admitted to the hospital following major trauma for the management of their injuries. Some of them need operation while other just need observation.

Admission criteria for ICU are cardiovascular resuscitation, airway protection and mechanical ventilation, invasive monitoring, severe head injury, organ support and correct coagulopathy.

### Discharge criteria

The discharge decisions of trauma patients differ between institutions and systems. However, below list are cover the general agreement about the patients who can discharge after trauma.

- Minor blunt trauma, hemodynamically stable on serial assessments.

- Negative e-FAST and CT scans for free fluid in body cavities.
- Minimal head injury with GCS 15 and normal CT scans without other body injuries and with normal neurologic status (with instructions to return for any changes in mental status, vomiting, or worsening headaches).
- Uncomplicated rib or sternal fractures.

## Referral

Multiple trauma patients who do not need admission for operation or observation are usually referred to a surgeon for the follow-up or management of their injuries.

**References and Further Reading**, click [here](#)



# Poisonings

by Harajeshwar Kohli and Ziad Kazzi

## Case

*An 18-year-old, previously healthy female, presents to the Emergency Department with nausea, vomiting, and tremors. She states 45 minutes ago she ingested an unknown number of diphenhydramine tablets (25 mg) in a suicidal gesture. Past Medical History: Depression, Medications: none. Social History: As per family member, she does not smoke or use illicit drugs. She is single and unemployed. Vital Signs: HR 110 bpm, BP 151/92 mmHg, RR 20 / min, Temp 38.5 degrees Celsius. Physical Exam: General Appearance: Mild distress, awake, appears to be hallucinating. Eyes: Dilated pupils bilaterally but reactive. Cardiovascular: Tachycardic, normal sounds, and no murmurs. Lungs: Clear to auscultation bilaterally. Abdomen: Soft, non-tender, non-distended, decreased bowel sounds. Neurologic: Normal motor power, normal cranial nerves, normal cerebellar exam, alert and*



Audio is available [here](#)

*oriented to self. Not oriented to location or date. Attention level waxes and wanes. Skin: warm, dry, no rash. Musculoskeletal: No deformities, no clonus, normal deep tendon reflexes.*

## Initial Approach

The initial approach to any patient presenting to the emergency department begins with airway, breathing, and circulation (the ABC's). The physician can proceed to a more thorough history and physical examination after the ABC's are secured. Oftentimes, patients who present after an overdose have altered mental status or try to conceal their ingestion. This highlights the need for the physician to gather collateral history from Emergency Medical Services (EMS) providers, bystanders, family, and friends. The physician should try to find out if the patient has any psychiatric history or access to medications. The physician should check the patient's clothing for empty bottles or paraphernalia of drugs of abuse. The physician should also inquire about the physical environment where the patient was initially found. Various environmental toxins can lead to altered mental status and should be considered in the initial assessment. For example, carbon monoxide released from



a defective space heater or electrical generator can cause altered mental status. Remember to always check a glucose level in an altered patient!

## Common Toxidromes

A toxidrome is a constellation of signs, symptoms and vital signs findings that clinically correlate with exposure to a toxin or class of toxins. The following list includes common toxidromes:

### Sympathomimetic (cocaine, amphetamines, phencyclidine)

- 👤 Hypertension
- 👤 Tachycardia
- 👤 Diaphoresis
- 👤 Mydriasis
- 👤 Agitation

### Anticholinergic (tricyclic antidepressants, diphenhydramine, antihistamines, jimson weed, atropine)

📌 Tachycardia

📌 Hyperthermia

📌 Dry skin

📌 Mydriasis

📌 Diminished bowel sounds

📌 Urinary retention

📌 Delirium, agitation

### **Cholinergic (organophosphates, carbamates, nerve agents) – SLUDGE<sup>B</sup>AM (mnemonic for muscarinic effects)**

📌 S- Salivation, seizure

📌 L- Lacrimation

📌 U- Urination

📌 D- Diarrhea

📌 G- GI distress (diarrhea and vomiting)

📌 E- Emesis

📌 B- Bronchorrhea

📌 A- Abdominal cramps

📌 M- Miosis

### **Cholinergic (organophosphates, carbamates, nerve agents) – MTWThF (mnemonic for nicotinic effects – days of the week)**

📌 M- Mydriasis

📌 T- Tachycardia

📌 W- Weakness

📌 TH- Hyperthermia

📌 F- Fasciculations

### **Opioid (opiates, opioids, clonidine)**

📌 Miosis

📌 Hypotension

📌 Bradypnea

📌 Bradycardia

📌 Hypothermia

📌 Depressed mental status

### **Sedative (benzodiazepines, gamma-hydroxybutyric acid)**

📌 Typically normal vital signs

📌 Depressed mental status

📌 Bradypnea

### **Vital Signs**

Vital signs can help guide the physician's differential diagnosis. The following table lists some toxins and their effect on vital signs:

**Table 3.23** Vital sign abnormalities and related toxins

iEM Table 1: Vital sign abnormalities and related toxins	
Vital Sign Abnormality	Toxin
Hyperthermia	Salicylates, Cocaine, Anticholinergics
Hypothermia	Opiates, Barbiturates, Sedatives
Hypertension	Cocaine, Amphetamines, Sympathomimetics
Hypotension	Beta Blockers and Calcium Channel Blockers
Tachycardia	Cocaine, Sympathomimetics
Bradycardia	Clonidine, Organophosphates, Beta Blockers
Tachypnea	Salicylates

*Produced by Harajeshwar Kohli and Ziad Kazzi*

## Physical Exam Findings

Physical exam findings can guide a physician's initial assessment of a possible overdose patient. Track marks could be a clue to intravenous drug abuse. The following table lists some key physical exam findings associated with certain toxins:

**Table 3.24** Physical exam findings and related toxins

iEM Table 2: Physical exam finding and related toxins	
Physical exam finding	Toxin
Miosis	Clonidine, Barbiturates, Opioids, Cholinergics, Pontine Stroke
Mydriasis	Cocaine, Sympathomimetics, Anticholinergics
Clonus and Rigidity	Neuroleptic Malignant Syndrome, Serotonin Syndrome, Depolarizing Neuromuscular Blockers
Dry Skin	Anticholinergics
Diaphoresis	Cholinergics, Hypoglycemia, Sympathomimetics
Cyanosis	Methemoglobinemia
Cherry Red Skin	Cyanide
Skin Blisters	Carbon Monoxide, Barbiturates

*Produced by Harajeshwar Kohli and Ziad Kazzi*

## Diagnostic Evaluation

The initial diagnostic workup for an overdose patient should be guided by clinical presentation and can be broad. Please note that this is an introductory chapter and the following is a basic initial approach and not meant to be exhaustive by any measure. As an initial suggestion, the following should be ordered initially:

- Complete Metabolic Panel (to assess electrolytes, anion gap, renal function, liver function)
- Complete Blood Count (to assess for hematologic disturbances)
- Serum acetaminophen (paracetamol) levels (which is a common cause of overdose, does not cause any clinical manifestations initially and can be lethal)
- Ethanol level and other drug levels based on history or suspicion
- Urine pregnancy test in any female of child-bearing age
- Serum salicylate levels are frequently obtained in overdose patients although salicylate toxicity can be suspected on initial clinical examination.
- Urine drug screens are not useful in the initial clinical assessment and management of overdose patients because

they are not able to detect a large number of drugs and can have false positives.

- For example, cocaine metabolites are detected for 3 days after use, synthetic opioids like fentanyl are not detected on routine urine drug screens, and pseudoephedrine produces a false positive screen for amphetamines.
- Urine drug screens are often requested for the psychiatric evaluation.

The anion gap is calculated as follows:  $Na - (HCO_3 + CL)$ , normal  $< 14$  typically. An elevated anion gap means there is an acidotic process going on and can support the diagnosis of a toxic ingestion. Many toxins and conditions can cause an elevated anion gap metabolic acidosis. The following table lists these substances and processes and the means by which they cause an anion gap acidosis (note the mnemonic CATMUDPILES[Table 3.25])

**Table 3.25** Causes of high anion-gap metabolic acidosis

iEM Table 3: Causes of high anion-gap metabolic acidosis	
Toxin	Comment
Cyanide	Lactic acid
Alcohol ketoacidosis	Acetoacetic acid
Toluene	Hippuric acid
Methanol	Formic acid
Uremia	Unknown
DKA	Acetoacetic acid/Beta Hydroxybutyric acid
Phenformin, Paraldehyde	Lactic acid
Iron, Isoniazid	Lactic acid
Lactic acid	Lactic acid
Ethylene glycol	Oxalic acid, Glycolic Acid, Glyoxylic acid
Salicylates	Salicylic acid

*Produced by Harajeshwar Kohli and Ziad Kazzi*

If a patient has altered mental status, always initially check a blood glucose level and consider empiric administration of dextrose, naloxone, and thiamine in the primary exam along with ABCs.. The physician should consider a non-contrast Computed Tomography scan of the brain to exclude intracranial pathology. An Arterial Blood Gas can be helpful to evaluate acid/base status, and co-oximetry can assess methemoglobin (MetHb) and carboxyhemoglobin (COHb) levels. If toxic alcohol ingestion is suspected, serum levels for methanol or ethylene glycol can be obtained but are not readily or rapidly available. Measured serum osmolality can be obtained to calculate the osmolar gap, which is the difference between the measured osmolality and calculated osmolality ( $= 2Na + BUN/2.8 + Glucose/18 + Ethanol/3.7$ ). A



normal osmol gap is between -12 and 10. An elevated osmol gap can be caused by methanol, acetone, ethanol, mannitol, sorbitol, isopropanol, lactic acid or ethylene glycol ingestion.

Additional diagnostic tests and imaging should be considered based on the history and presentation. For example, an EKG can be ordered to assess the impact of a certain drug on heart rate, rhythm and interval length.

A chest radiograph can be ordered to assess for pneumonitis after an ingestion or inhalation. An abdominal radiograph (KUB) can help identify radiopaque ingestions, including calcium carbonate, chloral hydrate, heavy metals, iron, phenothiazines, enteric coated and sustained released drugs (mnemonic CHIPES).

Always consider consulting a clinical toxicologist or a poison center for assistance with managing toxic overdoses.

## Common Treatments and Antidotes

A few common treatment modalities and **antidotes** will be outlined below.

### GI Decontamination

#### • Gastric lavage

- Within an hour of potentially lethal ingestion, particularly if no antidote treatment is available (i.e., colchicine, calcium channel or beta blocker)

#### • Charcoal

- Binds toxins in the stomach
- Does not bind metals, lithium, iron
- Should not be used in caustic ingestions and if there is a risk of vomiting and aspiration (altered mental status)
- The dose is 1g/kg with sorbitol or 25-50g in children
- Recommended within 1 hour of the ingestion

#### • Whole Bowel Irrigation

- Can be used with toxins that do not bind to charcoal (metals or lithium), and drug packets
- Recommended within 6 hours of ingestion
- Administer polyethylene glycol 1 liter/hour PO or per NG in adults and stop when rectal effluent is clear

#### • Multi-dose Activated Charcoal

- Useful for drugs that have enterohepatic and enteroenteric circulation (i.e., Digoxin, Theophylline, Carbamazepine)
- Useful for drugs with long gastrointestinal transit times, including sustained release products and drugs that impede GI motility (i.e., anticholinergics, opiates)
- Dose is 25 g every 4-6 hours for 2-3 doses

## • Enhanced Elimination

- Urinary alkalinization
  - Enhances excretion of weak acids
  - Recommended for salicylic acid and phenobarbital overdoses
- Hemodialysis
  - Can be used for ethylene glycol, methanol, isopropyl alcohol, salicylic acid, and lithium. Best with small-sized substances that are not protein bound and that have a small volume of distribution.

References and Further Reading, click [here](#)

**Table 3.26** Common Toxins and Antidotes

iEM Table 4: Common toxins and antidotes	
Toxin	Antidote
Acetaminophen	N-acetylcysteine
Methanol/Ethylene Glycol	Ethanol/Fomepizole
Iron	Deferoxamine
Opioids	Naloxone
Anticholinergics	Physostigmine
Carbon Monoxide	100 Percent Oxygen, Hyperbaric Oxygen
Organophosphates	Atropine/Pralidoxime
Methemoglobinemia	Methylene Blue
Cyanide	Hydroxocobalamin, Sodium Nitrite, Sodium Thiosulfate
Digoxin	Digoxin Immune Fragments
Beta Blockers	Glucagon, High Dose Insulin
Calcium Channel Blockers	High Dose Insulin
Oral Hypoglycemics	Glucose, Octreotide

*Produced by Harajeshwar Kohli and Ziad Kazzi*

# Respiratory Distress

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by Ebru Unal Akoglu

## Case Presentation

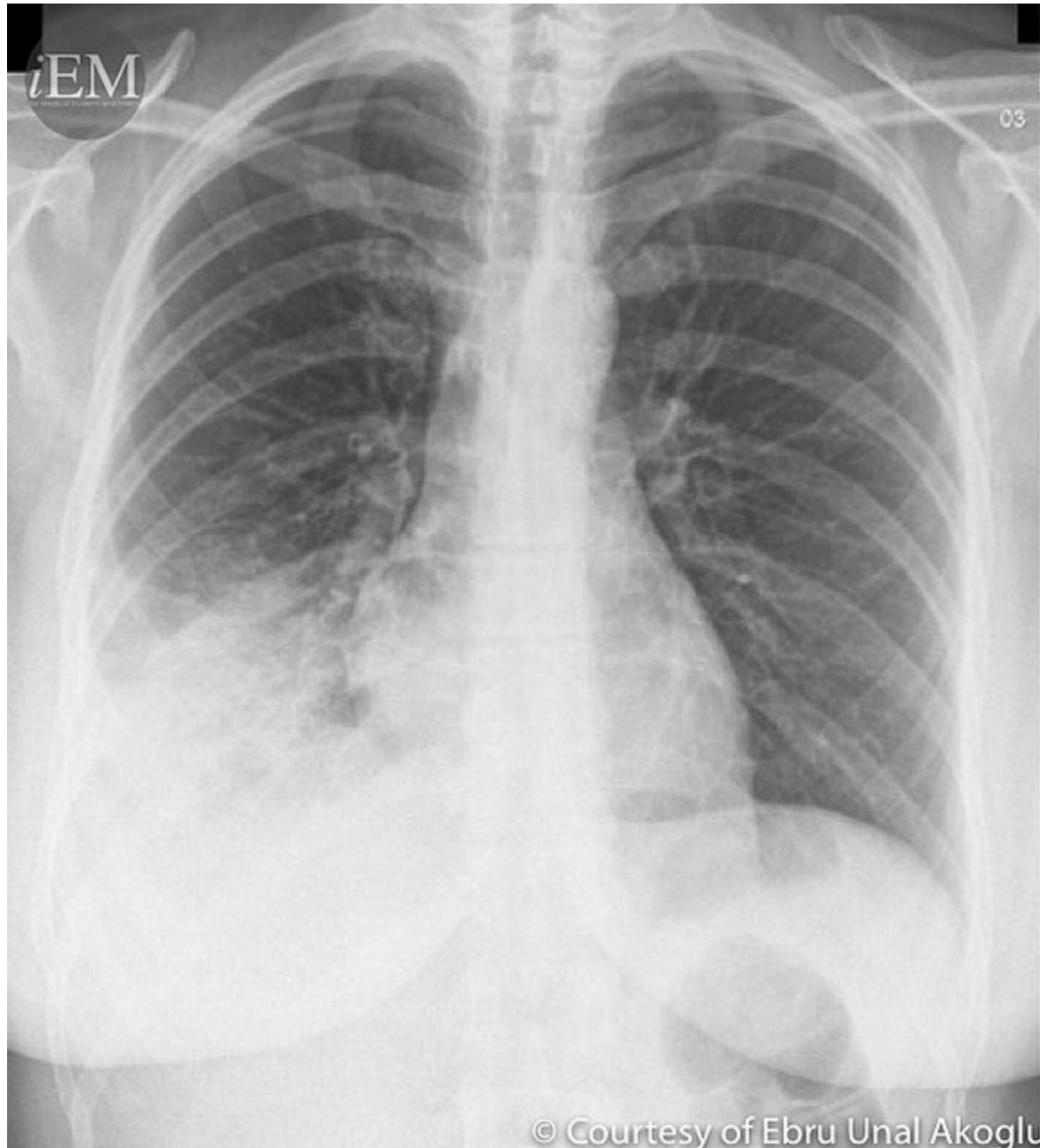
*A 40-year-old female with a history of diabetes mellitus presents with a complaint of 6 days cough and muscle aches. Patient has right-sided chest pain with deep breathing. Her vitals are the following: temperature 37.1 degrees Celcius; blood pressure 150/97 mmHg; heart rate 120 bpm; respiratory rate 19/min; and pulse oximetry 89%. On physical examination, she has diminished breath sounds and ronchi at the right bases. Her chest X-ray is shown in Image 3.18.*



Audio is available [here](#)

What are the diagnostic considerations? What is your next move?  
What is the most appropriate management strategy?

**Image 3.18** Chest x-ray.



In the emergency department, respiratory distress is a challenging chief complaint and diagnosis, and you should evaluate, examine and ease (treat) the patient simultaneously. You have to act quickly with limited information, or your patient can decompensate in front of you.

Objectives of this chapter are listing the causes of respiratory distress, describing the initial approach to a patient with respiratory distress and discussing the initial management plan for a patient with respiratory distress.

## Introduction

Respiratory emergencies are common presentations to emergency departments. Appropriate assessment and timely interventions may be crucial in dyspneic patients. Respiratory distress is responsible for nearly 4 million ED visits each year and is one of the most common presenting complaints in the elderly. Management of acute respiratory distress is a challenging task. Good patient outcomes rely on your ability to assess ventilation, oxygenation, work of breathing, lung function, airway resistance and air flow.

When a patient presents with dyspnea, the primary task of the emergency physician is to assess for and ensure the stability of the patient's airway, breathing, and circulation (ABC).

Respiratory distress is used to describe varying degrees of problems in the respiratory system.



Rapid assessment may necessitate intubation, BiPAP (Bilevel Positive Airway Pressure), nebulizations, decompression or other therapies in the immediate period following the patient's arrival. Sometimes, it may be hard to decide whether your patient needs medication, suctioning, airway management, intubation, mechanical ventilation support (invasive, non-invasive) or just close observation.

Respiratory distress is a term utilized to summarize a complex of clinical features. These are tachypnea, hypoxemia (peripheral arterial oxygen saturation [SpO2] <90% on room air), increased work of breathing (intercostal, subcostal, or suprasternal retractions; nasal flaring; grunting; use of accessory muscles) apnea, altered mental status, and cyanosis which is characterized by >5gr/dL of deoxygenated hemoglobin. At this moment, it is better to share some terms and definitions. Please check Table 3.27.

**Table 3.27** Terms and definitions in respiratory distress

iEM	Table 1: Terms and Definitions in Respiratory Distress
<b>Dyspnea</b> is a subjective feeling of difficulty in breathing. It is experienced and described differently depending on the cause.	
<b>Tachypnea</b> is an increase in the rate of respiration.	
<b>Hyperpnea</b> is an increase in depth of breathing, not always accompanied by tachypnea.	
<b>Hyperventilation</b> is a sensation of dyspnea associated with excessive breathing.	
<b>Orthopnea</b> is a difficulty in breathing except when in an upright position.	
<b>Paroxysmal nocturnal dyspnea</b> is a sudden attack of dyspnea that usually occurs when patients are sleep. The patients awaken gasping for air and try to sit up to relieve the symptoms.	

by Ebru Unal Akaglu

Healthy lungs are the cornerstone of fluid regulation among the interstitium and alveoli, which can be destroyed by lung injury.

Lung injury can cause abnormal gas exchange, impaired compliance, and pulmonary pressure. Normal lung function requires dry, patent alveoli assisted by proper capillary perfusion and patent endothelium.

Respiratory distress is a consequence of an alveolar injury producing diffuse alveolar damage. Tumor necrosis factor, interleukin (IL)-1, IL-6, and IL-8, are the pro-inflammatory cytokines released after injury and recruit neutrophils to the lungs. Activation of neutrophils causes endothelium damage that ends with impairment of hydrostatic and oncotic forces of membranes.

Damage to the capillary endothelium causes the escape of proteins from intravascular space. The membranous hydrostatic and oncotic forces are lost, and the interstitial space fills with fluid. Also, the clearance ability of the membranes may be lost.

Increase in interstitial fluid, combined with damage to the alveolar epithelium, causes the air spaces to fill with bloody, proteinaceous edema fluid and debris from degenerating cells. Besides, the functional surfactant is lost, resulting in alveolar collapse.

Lung injury has numerous consequences including impairment of gas exchange, decreased lung compliance, and increased pulmonary arterial pressure.



Patients with acute respiratory distress tend to progress through three relatively discrete pathologic stages. These are 1) Exudative stage: diffuse alveolar damage, 2) Proliferative stage: resolution of edema, squamous metaplasia, deposition of collagen, and 3) Fibrotic stage: diffuse fibrosis and cyst formation.

Acute respiratory distress is the clinical consequence of lung injury. Many predisposing factors may lead to lung injury (Table 3.28). Associated abnormalities increase the risk for adverse outcomes.

### Initial Stabilization

The following three assessment questions guide management:

**Table 3.28** Predisposing factors

iEM	Table 2: Predisposing Factors for Respiratory Distress
Aspiration Drug and alcohol Genetic determinants Lung and stem cell transplantation Massive transfusion Others (acute pancreatitis, cardiopulmonary bypass, drowning, obesity, pneumonectomy, smoking, thoracic surgery) Pneumonia Sepsis Severe trauma	

Adopted from UpToDate, please check the link in references to see more details. by Ebru Unal Akoglu

1. Is the airway patent? (A)
2. How adequate is breathing? (B)
3. Is oxygenation sufficient? (C)

### Airway

Abnormal breath sounds often point to the obstruction. Snoring indicates obstruction of the airway, usually by the tongue. Simple interventions can lead to marked improvement. For example, head tilt maneuver or a nasopharyngeal/oropharyngeal airway often eliminates snoring. Inspiratory stridor suggests obstruction above the vocal cords (a foreign body obstruction or epiglottitis). Any foreign body should be removed immediately. Expiratory stridor often comes from below the cords (as in croup or a deeper foreign body).

### Breathing

Coarse lung sounds, formerly called rhonchi, generally result from secretions in the airway. Nasotracheal suctioning of accumulated secretions using a soft, flexible catheter clears coarse-sounding lungs. Wheezing suggests flow restriction below the level of the trachea, whereas crackles (or rales) indicate the presence of fluid or atelectasis at the alveolar level. Administration of an inhaled bronchodilator significantly reduces wheezing.

The most difficult management part of a patient with respiratory distress is ventilation support. Unfortunately, unrecognized inadequate breathing, failure of ventilator support or unassured airway will ultimately lead to cardiopulmonary arrest. If breathing is inadequate, ventilation must be provided immediately. Ventilation can be non-invasive or invasive.

Non-invasive refers to ventilator support provided through the patient's upper airway, usually using an oxygen mask, nasal cannula or bag-valve-mask (BVM) depends on the patient's need.

Invasive refers to ventilator support provided by passing the upper airway with an endotracheal tube, supraglottic airway (e.g., laryngeal mask airway or laryngeal tube) or tracheostomy depending on the need. Unassured airway patency is the most important indication for invasive ventilation.

## Circulation

Hypoxia is the lack of sufficient oxygen in the body. Hypoxia may result from an airway patency problem, failure of ventilation support, or an intact airway with good breathing but poor perfusion and oxygenation. In airway management, the first approach is the clearance of airway and positioning; this maneuver often increases oxygen saturation and improves ventilation. If oxygen saturation does not increase, the second approach

is oxygen support using an oxygen mask, nasal cannula or bag-valve mask, depending on the patient's need. If oxygen saturation still does not increase, airway devices should be applied, or the patient should be intubated. The aim is the correction of hypoxia ideally to maintain saturation at 94-98% by titration of oxygen carefully. The treatment depends on the condition that causes respiratory distress. However, in a general approach, CAB+D – circulation, airway, breathing, and drugs is the perfect treatment protocol in the ED.

Some critical actions should be done at every stage if necessary.

C – Circulation: two large bore IV access, fluids if hypotensive, monitorization

A – Airway: oropharyngeal or nasopharyngeal airway devices, LMA, some maneuvers (Head Tilt, Heimlich), suction, medication for an allergic reaction

B – Breathing: Oxygenation (nasal cannula, non-rebreather mask) if not responding, next step is non-invasive mechanical ventilation support or intubation

D – Drugs: Depending on the patient's primary problem causing respiratory distress, appropriate drugs should be implemented to management accordingly.

The three signs of impending respiratory arrest are:

- Decreased level of consciousness;
- Inability to maintain respiratory effort;
- Cyanosis.

Presence of one or more of these needs immediate intervention. The untreated respiratory arrest will lead to cardiac arrest eventually. Life-threatening conditions, such as airway obstruction, acute coronary syndrome, pneumonia, cardiac tamponade, pulmonary embolism, asthma, anaphylaxis, trauma,

and exacerbation of chronic obstructive pulmonary disorder may lead to respiratory distress and arrest. These critical problems should be treated during the assessment.

While you are examining the patient, other staff members (such as intern, nurse, paramedic) may measure vital parameters and monitor the patient, obtain intravenous access and do ECG. Teamwork will accelerate your assessment process and allow you to formulate a treatment plan while others obtain a history from family or friends.

## History Taking and Physical Examination Hints

### History Taking Hints

Acute respiratory distress is one of the most common chief complaints in the ED. The differential diagnosis includes many disorders, so a careful history can be helpful to narrow this wide differential. In addition, past medical and family history, trauma, travel, medications, allergies and exposures should be considered with

common symptoms. Not only family members, but also a brief conversation with the paramedics, who transferred the patient, can give you useful information about the patient and the surrounding area they took the patient from.

### Physical Examination Hints

Although we do a focused and goal-directed physical exam in critical patients, a detailed physical examination also provides important guidance.

The general appearance of patient – confusion, cyanosis, drowsiness, tachypnea, and pallor – can guide your management. Also, respiratory rate and oxygen saturation are two vital sign measurements that are helpful in assessing and monitoring the degree of respiratory distress. The higher the respiratory rate, the greater the work of breathing and the more likely the patient will eventually get tired. Oxygen saturation is important not only in assessing but also following the progress of the patient.

Pulse oximetry is a valuable monitoring tool for the management of respiratory distress patients. It is useful for either making a decision when to administer oxygen or titration of oxygen to avoid patient harm from too much oxygen.

For respiratory distress patients without immediate life threats, your next assessment focus should be to determine the patient's work of breathing and respiratory pattern (video), looking for any tripodding or retractions. Retractions can be visualized during the assessment of chest movements, and they are more valuable than lung sounds in the decision of the respiratory distress severity.

Lung sounds (video) such as wheezing, rales, ronchi, and stridor further guide the differential diagnosis. Decreased sounds or hyperresonance may also provide additional clues. Lung sounds should be examined from both sides of the chest wall even in supine positioned patients (video). Orthopnea, or the inability to lie

flat, is not a test, but rather, a question to ask the patient. Sweating and diaphoresis in an environment where others are not sweating, suggests significant distress.

Jugular venous distension (picture), S3 gallop, and peripheral edema indicate that the patient has fluid overload. Heart sounds such as murmur, or decreased sounds, guide the differential and also management. Pulses must be assessed bilaterally.

It is important to remember that anxiety is common in patients with significant medical problems, just as in trauma. COPD patients have it more often than the general population. Secondly, even healthy, young patients may have a medical cause for hyperventilation. A thorough assessment is important not to miss clues of a medical or traumatic condition.

Key findings of severe respiratory distress are 1) retractions and use of accessory muscles, 2) inability speak full sentences, 3) inability lie flat, 4) extreme diaphoresis,

5) restlessness, agitation, decreased level of consciousness.

## Differential diagnosis

Having a wide differential diagnosis list for respiratory distress will allow you to sort through the possible causes more rapidly. In the ED, you must think the worst case scenarios first, and you should try to rule out them. Respiratory distress differential diagnoses list has various critical diseases. These are anaphylaxis, asthma/COPD, acute coronary syndrome, pulmonary edema, pulmonary embolism, pneumonia, pericardial tamponade, tension pneumothorax, and upper airway obstruction.

The above diagnoses are crucial and should be treated immediately. Other causes of respiratory distress should also be assessed and managed properly.

## Emergency Diagnostic Tests and Interpretation

Multiple tests are available to narrow the differential diagnosis of respiratory

distress. Generally, laboratory and radiological tests take a long time; you should start the treatment before getting results.

## Bedside tests

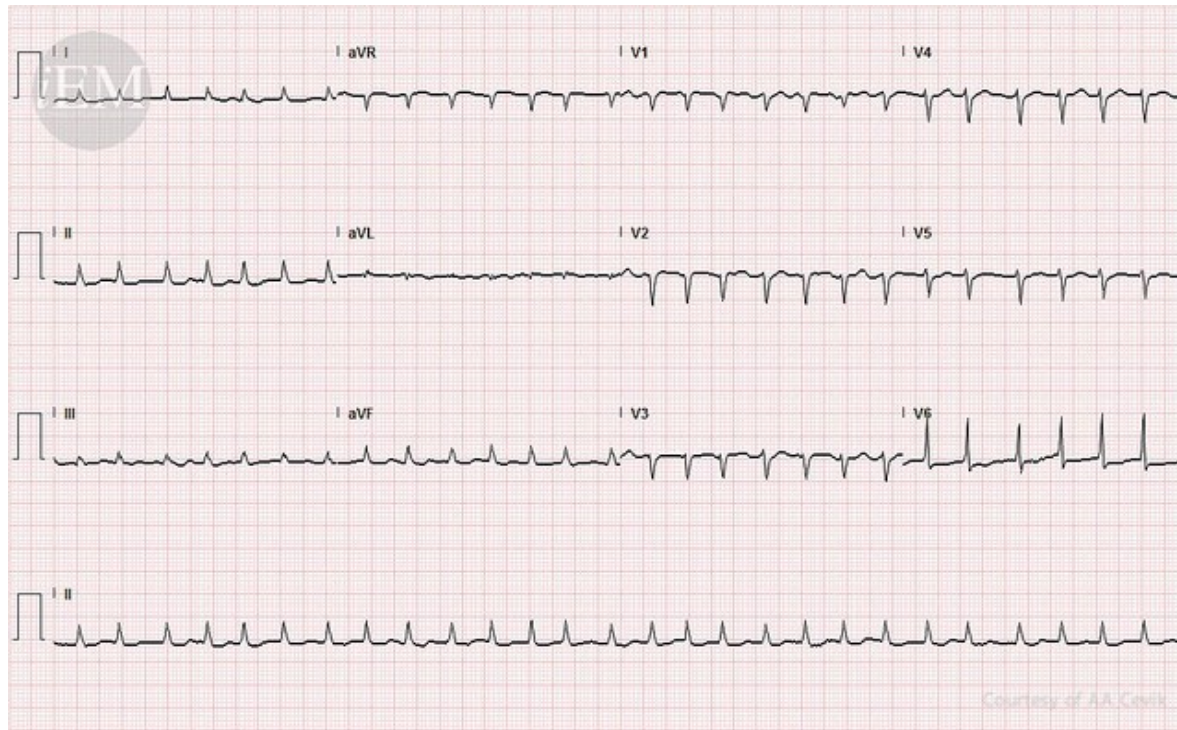
- ECG, especially in elderly patients who usually present atypically with dyspnea in acute coronary syndrome, is easy and practical.

What are your diagnosis and next action about the ECG in a patient with shortness of breath and palpitation (Image 3.19)? - Case – 68 yo female presented with palpitation, dyspnea, unable to lay down. Vitals are BP: 80/43 mmHg, HR: 160 bpm, RR: 32 pm, Temp: 37 Celsius, SatO2: 87%. Patient diaphoretic, cool, anxious. Chest auscultation revealed basal to mid zone crackles on both sides. Heart sound irregular. Bilateral 1+ pitting edema.

- Bedside glucose level should be obtained in cases of a decreased level of consciousness and suspected metabolic acidosis.



### Image 3.19



Case – 68 yo female presented with palpitation, dyspnea, unable to lay down. Vitals are BP: 80/43 mmHg, HR: 160 bpm, RR: 32 pm, Temp: 37 Celsius, SatO2: 87%. Patient diaphoretic, cool, anxious. Chest auscultation revealed basal to mid zone crackles on both sides. Heart sound irregular. Bilateral 1+ pitting edema.

### Laboratory tests

- Arterial blood gas analysis is useful, quick and important to determine metabolic and/or respiratory cause of respiratory distress.
- Besides these, complete blood count (CBC), troponin, renal panel, BNP, and D-dimer can be used to assess differential diagnosis of respiratory distress.

### Imaging

- Chest X-ray and Computed Tomography are generally indicated to detect and differentiate pathologies. Pneumothorax, pneumonia, pulmonary embolism, pleural effusion, cardiac tamponade, etc. are the important causes of respiratory distress.

What are your diagnoses about the chest x-rays (Images 3.20 and 3.21) in patients with shortness of breath?

### Image 3.20

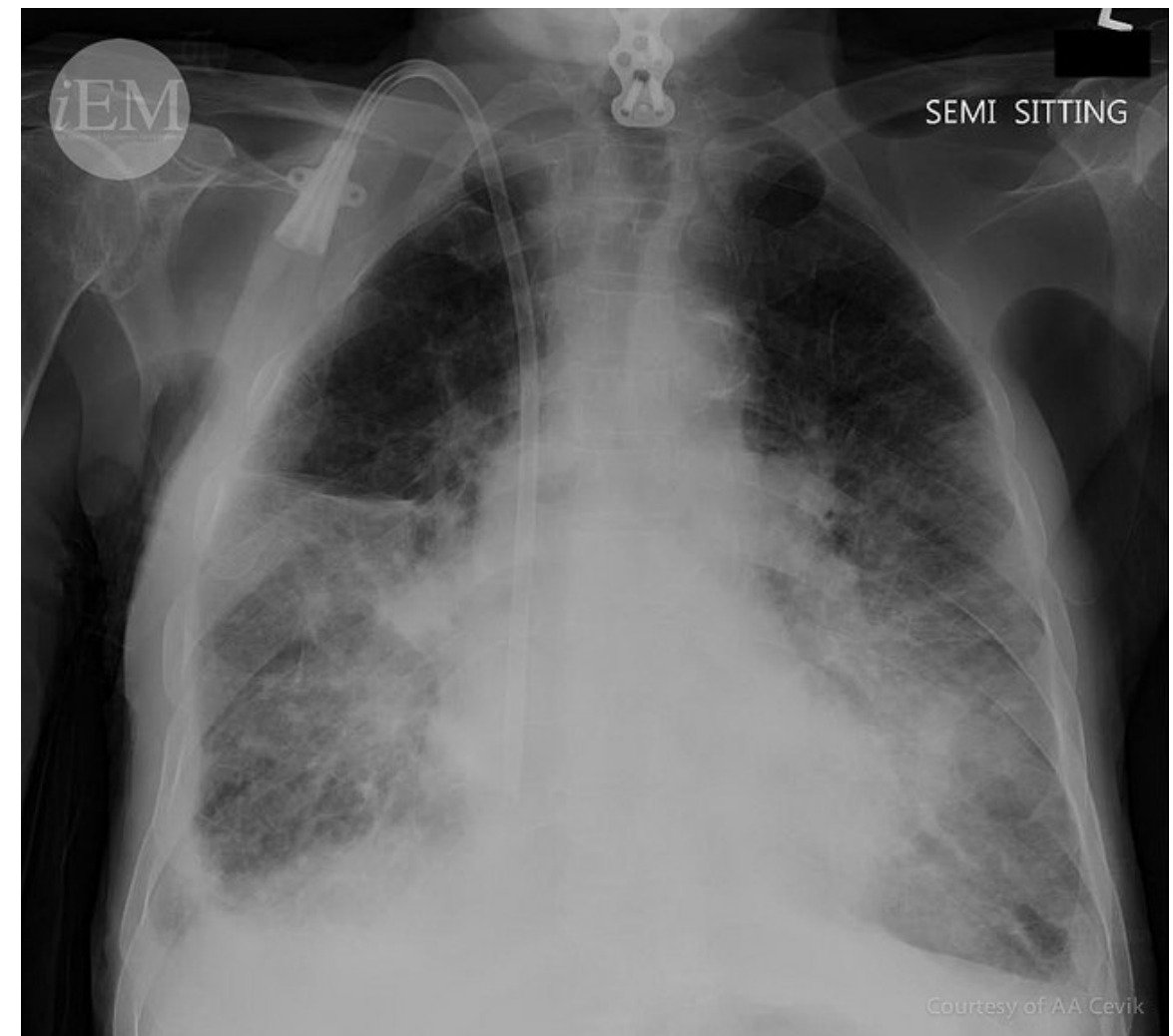
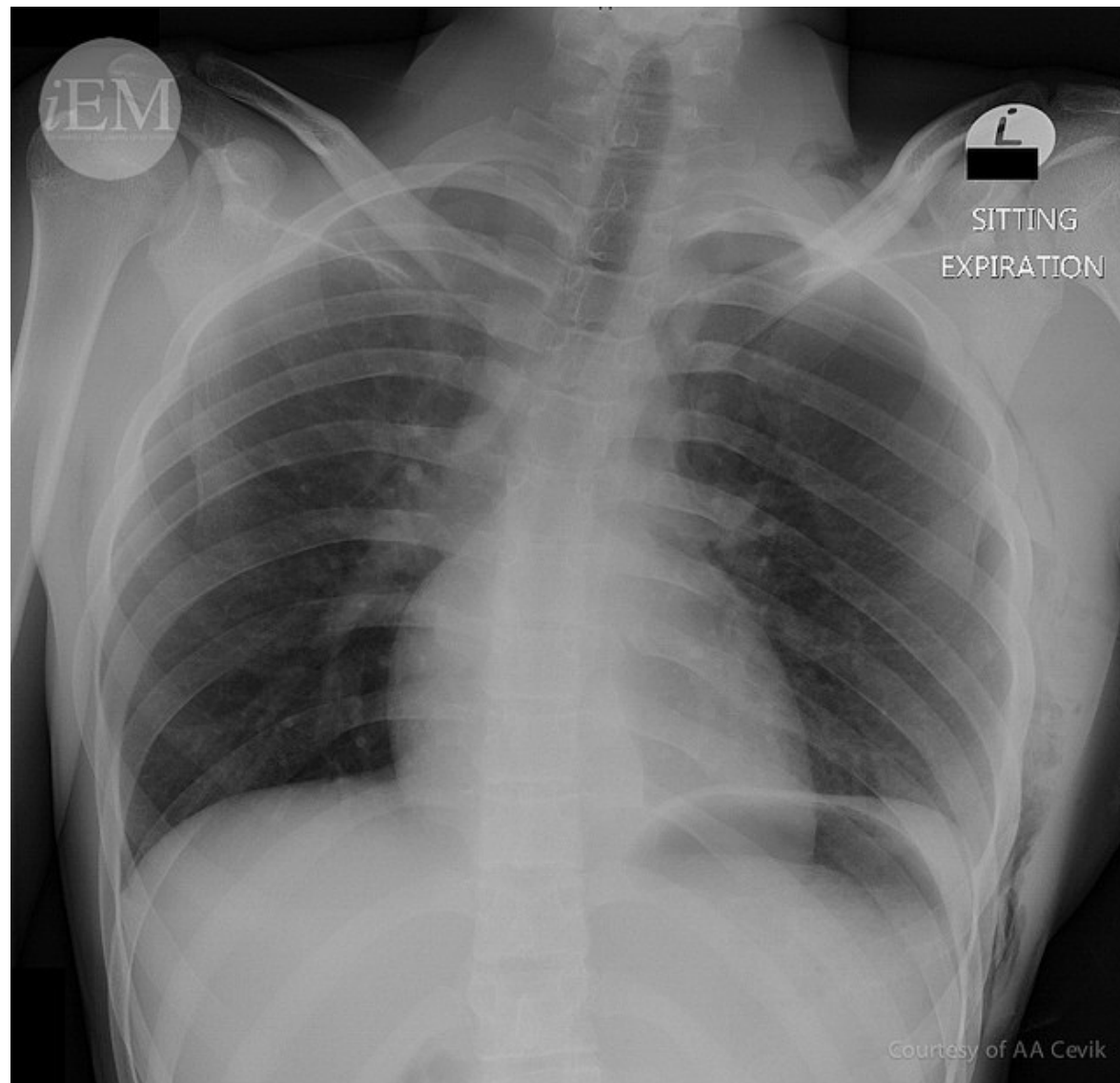




Image 3.21



- Currently, ultrasonography is another option in the evaluation of respiratory distress (watch this [video](#)). Ultrasonography provides valuable information about the origin of symptoms and often diagnosis in the initial assessment of the patient. Also, ultrasonography is faster than laboratory tests and other imaging modalities, repeatable, and portable so that it can be used for unstable patients. It is also cost-effective.

## Emergency Treatment Options

Because of a variety of diseases can cause respiratory distress and specific diseases need specific approaches such as tension pneumothorax needs decompression of the air from the chest; asthma and COPD need bronchodilator treatments. Please check each critical diagnosis separately in other chapters.

Patients often die from the complications of respiratory distress. The initial assessment is crucial, and essential interventions should be made immediately.

Respiratory distress symptoms usually have a wide range of ineffective breathing or respiratory arrest and difficulty in speaking, accompanied by cyanosis and diaphoresis.

Immediate assessment priorities for any difficulty breathing include quick determination of circulation, airway, and breathing as described above.

## Disposition

if the patient's condition or blood gas analyze does not improve despite therapy admission should be considered to appropriate clinics.

**References and Further Reading**, click [here](#)

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by Maryam AlBadwawi

## Case presentation

*A 61-year-old male with fever, shortness of breath and vomiting was brought to the ED by ambulance. He also complained of dizziness, malaise, and reduced urination. His symptoms started one week earlier and got progressively worse in time. However, he did not seek any medical assistance before. His medical history includes diabetes mellitus and hypertension.*

*On examination, he appeared ill. His vital signs were: BP: 80/50 mmHg, HR: 140 bpm, T: 38.6°C, RR: 30 bpm, SPO2: 90% on room air. His blood sugar was 5.3 mmol/dL, and capillary refill was four seconds. He responds to verbal comments on the AVPU score. On auscultation, coarse crackles were heard on the right side. Heart sounds were normal. There was 1+ pitting edema on bilateral legs.*



Audio is available [here](#)

*Abdominal examination was unremarkable, and there was no focal neurological deficit.*

*The pre-diagnosis was the septic shock. He needed emergent resuscitation and antibiotics administration.*

*Two large bore IV cannulas were inserted. CBC, urea, electrolytes, creatinine, LFT, cardiac enzymes, lactate, and ABG were ordered. Bedside US scan revealed a collapsing inferior vena cava. A urinary catheter was placed for urinalysis and to monitor the output.*

*While waiting for the results, one liter of isotonic saline, one dose of Vancomycin (15-20 mg/kg) and Piperacillin-Tazobactam ( 80-100 mg/kg) were administered. His BP did not improve. After the second liter of isotonic saline, his HR settled to 120, but his mean arterial pressure (MAP) remained below 60 mmHg. A central line was inserted, and epinephrine (2-10 mcg/min) started.*

*His MAP improved to 65 and remained stable. He was transferred to the ICU.*

## Introduction

Shock, in simple terms, is a reduced circulatory blood flow state within the body. The inadequate circulation deprives the tissues of its oxygen and essential nutrients. Mitochondria are the first cellular structure to be affected by hypoxia. As a result of anaerobic respiration, they start to produce lactate, leading to lactic acidosis. Initially, the shock is reversible. Extended shock state leads to cellular and organ dysfunction and ultimately death.

The shock is a life-threatening medical emergency with a mortality rate up to 20%. The primary goal of resuscitation is to optimize organ perfusion. It may result from a variety of causes. It is divided into four categories according to the mechanism of the disease and treatment (Table 3.29). Differentiating the type facilitates treatment, however, all categories of shock lead to the same result.

**Table 3.29** Shock types

SHOCK TYPE	PHYSIOLOGY	MECHANISM/ CAUSES	CLINICAL EXAMPLES
Hypovolemic	The decrease in plasma or RBC + plasma	External bleeding Internal bleeding  Third spacing loss, GI, renal, or insensible losses (hemorrhagic)	Trauma, AAA rupture, ectopic pregnancy rupture, nausea and vomiting, inadequate intake, renal diuresis, paraesthesia, burns.
Cardiogenic	A primary cardiac problem decreasing cardiac output.	Pump failure from myocardial injury or dysfunction Arrhythmias Valvular disease Ventricular septal defect	Ischemia and infarct, Myocarditis, Cardiomyopathy VT, VF Aortic regurgitation
Obstructive	Non-cardiac obstruction affecting cardiac filling or emptying	Impaired RV diastolic filling Impaired RV filling due to obstructed venous return Increased right ventricular afterload Increased left ventricular afterload	Cardiac tamponade, Constrictive pericarditis Tension pneumothorax Pulmonary embolism Aortic dissection
Distributive	Extreme peripheral vasodilation	Peripheral pooling Capillary leak Myocardial depression	Sepsis or SIRS Anaphylaxis Neurogenic shock Rewarming in severe hypothermia Endocrinological

Adopted from following references. Please read (Marx, J. A., Hockberger, R. S., & Rosen, P. (2014). *Rosen's emergency medicine: Concepts and clinical practice* (8th ed., Vol. 1). Philadelphia, PA: Mosby Elsevier and Avegno, J. CDEM Self-Study Modules. *The approach to shock*. Retrieved May 11, 2016) references to get more information.



## Critical Bedside Actions and General Approach

Early and accurate management is essential as it reduces mortality significantly in certain types of shock. Heart rate, blood pressure, and partial oxygen saturation must be continuously monitored. Optimizing airway, breathing, and circulation (ABC) is the priority. Check the airway of the patient, and consider intubation in case of inadequate oxygenation and ventilation. Maximizing arterial oxygen saturation through proper oxygenation is crucial. Central venous oxygenation should be monitored with a target of minimum 70%. Reducing the work of breathing lessens the metabolic load.

## History and Physical Examination Hints

Common features of the shock include hypotension, altered mental status, and oliguria, regardless of the etiology. The patient history is significant to diagnose the type of shock and accurately treat the patient.

- Hypovolemic shock might have a history of trauma, pregnancy, gastrointestinal losses or burn. Initially, heart rate and force of contraction increase. Vasoconstriction causes elevated diastolic BP, and pulse pressure (the difference between systolic and diastolic BP) narrows. The blood flow to the noncritical organs decreases so that cells produce lactic acid. As bleeding continues, ventricular filling and cardiac output (CO) decrease, resulting in decreased BP. Hypotension is a late sign of shock.

- Cardiogenic shock occurs when more than 40% of the myocardium undergoes necrosis from ischemia, inflammation, toxins or immune destruction. It induces the same impairment as hemorrhagic shock. Patients have evidence of ventricular dysfunction earlier in the disease.
- Obstructive shock should be considered in patients with chest pain, shortness of breath, and altered mental status. The physical examination may reveal jugular venous distention, muffled heart sounds, pulsus paradoxus, tachypnea, tachycardia, cold extremities, friction rub, new murmur, and signs of deep vein thrombosis.
- Distributive shock findings depend on the cause. Sepsis and septic shock cause signs of infection. Other symptoms and signs include hypo/hyperthermia, tachycardia, tachypnea, wide pulse pressure, warm extremities, altered mental status, oliguria, and skin rash. Anaphylactic shock is characterized by skin and mucosa manifestation such as urticaria, flushing, pruritis, and angioedema. Respiratory symptoms may include rhinitis, bronchospasm, dyspnea, and stridor (pharyngeal/laryngeal edema). The patient may experience dysrhythmias, hypotension, presyncope, and syncope. Additionally, GI symptoms such as nausea, vomiting, and diarrhea may be present.
- Blood pressure may be normal or even high in the early course of shock; however, if left untreated, it may proceed to



tachycardia and hypotension. Shock Index (heart rate divided by systolic blood pressure), may reveal obscure shock. The normal shock index ranges from 0.5 to 0.7. A value of  $>0.9$  is considered abnormal and associated with higher mortality.

## Emergency Diagnostic Tests and Interpretation

The suspected cause of shock, attributed from the history and physical examination, should guide diagnostic testing. The goal is to determine the involvement of organ hypoperfusion and damage. The following are helpful investigations in shock:

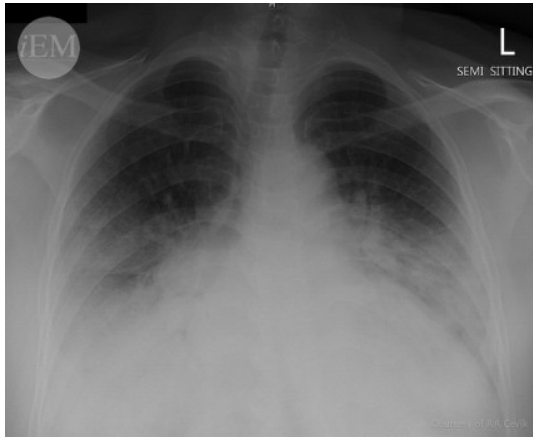
- **Complete blood count** and coagulation profile
  - Anemia, infection, hypo-coagulopathy related abnormalities can be seen
- Electrolytes
  - Some of the cases may show electrolyte disturbance because of their comorbidities or continuous medical problems affecting their nutrition or metabolism.
- Renal function tests
  - Blood urea nitrogen/creatinine
    - Renal functions impair because of low perfusion. High blood urea nitrogen compared to creatinine may provide

information about prerenal causes showing a volume or blood loss.

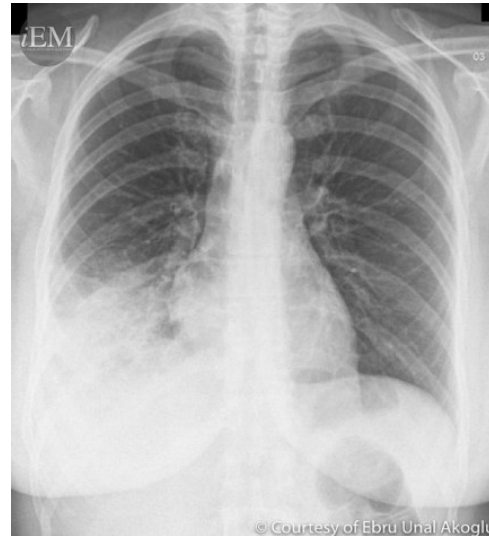
- Urinalysis
  - Urosepsis is one of the common sepsis causes.
- Hepatic function tests
  - Hepatic functions impair because of low perfusion.
- Lactate
  - Lactate gives an opinion about the hypoperfusion status. Its levels considered normal between 0.5-1 mmol/L. Lactate levels more than 2 considered as abnormal in the critically ill patients. Levels more than 4 shows increased the risk of mortality and morbidity. Therefore, those levels are used for some institutions to decide ICU admission.
- Urine pregnancy test
  - Considering every female patient in childbearing age as a pregnant patient is essential thinking in the ED.
- **Chest x-ray**
  - For pneumonia, pleural effusion, and other possible shock causes such as cardiac tamponade, aortic dissection, pneumothorax (tension).

What are your diagnoses in hypotensive patients with below chest x-rays?

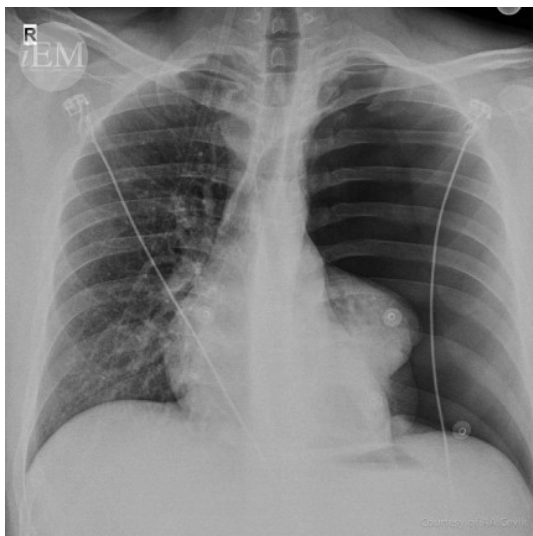
**Image 3.22**



**Image 3.24**



**Image 3.23**



- ECG
  - For arrhythmias, MI, cardiomyopathy and other findings
- US (**RUSH** protocol to find the cause of the shock (**video**))
- Other invasive tests:
  - Arterial blood gas analysis for O2 pressure and pH level,
  - Some centers may prefer to measure systemic vascular resistance, central venous oxygen, and cardiac output in the ED.
- Certain etiologies of shock will require additional investigations:
  - SIRS and sepsis: Cultures (blood, sputum, urine, or wound), head CT and lumbar puncture
  - Cardiogenic: ECG, cardiac enzymes, and echocardiography
  - Obstructive: CT or V/Q scan for PE, echocardiography for cardiac tamponade

**Table 3.30** Important Physiological Changes in Shock

SHOCK TYPE	HEART RATE	CENTRAL VENOUS PRESSURE	CONTRACTILITY	EXTREMITIES	SYSTEMIC VASCULAR RESISTANCE	TISSUE PERFUSION/ SCVO <sub>2</sub>
Hypovolemic	Increased	Decreased	+/- Increased	Cool	Increased	Decreased
Cardiogenic	Increased	Increased	Decreased	Cool	Increased	Decreased
Obstructive	Increased	+/- Increased	+/-	Cool	Increased in Tamponade and PE, but decreased in tension pneumothorax	Decreased or Increased
Distributive	Increased	Decreased	+/-	Warm	Decreased	Decreased

In any ill-appearing patient with tachycardia and hypotension or high shock index, the shock must be considered. The mentioned signs, symptoms and relevant diagnostic tests often help to arrive at a diagnosis and initiate appropriate treatment. However, certain disease processes can complicate the picture and lead to an alternate diagnosis. Therefore, understanding the shock physiology is important (Table 3.30). The labs should be completed, but it is important not to wait for the results before initiating treatment.

Using certain criteria to help make the diagnosis and point to specific types of shock is more beneficial.

## Septic shock

- SIRS (Systemic Immun Response Syndrome) – Two or more of the following:

1. Temperature  $>38^{\circ}\text{C}$  or  $<36^{\circ}\text{C}$
2. Heart rate  $>90$  beats/min
3. Respiratory rate  $>20$  breaths/min or  $\text{PaCO}_2 <32$  mmHg
4. WBC  $>12,000/\text{mm}^3$ ,  $<4,000/\text{mm}^3$ , or  $>10\%$  band neutrophilia

- Sepsis

- SIRS with finding the source of infection and associated with organ damage or hypoperfusion.
- Sequential Organ Failure Assessment (S.O.F.A. or sofa) was recently described and created multiple discussions in emergency and critical care journals. q (Quick) sofa score includes Hypotension: systolic blood pressure less than or equal to 100 mmHg, Altered mental status, and

Tachypnoea: respiratory rate greater than or equal to 22 breath per minute. 2 or more criteria violation in Q sofa score is considered the poor outcome predictor.

- Septic shock

- Sepsis and hypotension despite adequate fluid resuscitation.

## Hemorrhagic shock

- Simple hemorrhage

- Suspected bleeding with normal vitals and normal base deficit

- Hemorrhage with hypoperfusion

- Suspected bleeding with base deficit  $<-4$  mEq/L or persistent pulse  $>100$  beats/min

- Hemorrhagic shock

- Suspected bleeding with at least four of the following criteria:

1. Ill appearance or altered mental status

2. Heart rate  $>100$  beats/min

3. Respiratory rate  $>20$  breaths/min or  $\text{PaCO}_2 <32$  mmHg

4. Arterial base deficit  $<-4$  mEq/L or lactate  $>4$  mmol/L

5. Urine output  $<0.5$  mL/kg/hr

6. Arterial hypotension  $> 30$  continuous minutes

## Cardiogenic shock

- Cardiac failure

- Clinical evidence of impaired forward flow of heart, including presence of dyspnea, tachycardia, pulmonary edema, peripheral edema, or cyanosis.

- Cardiogenic shock

- Cardiac failure and at least four criteria of that similar to hemorrhagic shock.

## Emergency Treatment Options

### Fluid Resuscitation

Two large bore IV access should be obtained to support the circulatory system. A central line is also very beneficial in delivering fluid and medication, especially inotropes. Crystalloid fluids (normal saline or Ringer's lactate) should be used in boluses (2-3 L bolus in 5-20 min – 20ml/kg in neonates and pediatrics). Pay close attention to patients in cardiogenic shock. Do not administer I.V. fluids rapidly to patients with signs of pulmonary congestion. Small fluid boluses such as 250 mL should be preferred in those cases.

### Blood Products Resuscitation

Blood transfusion is considered if there is no response to two liters of fluid boluses, ongoing hemorrhage, or impending cardiovascular collapse. O-negative blood is standard for child-bearing women and O-positive in men is

acceptable. Additionally, controlling the source of bleeding is critical. In special cases like hereditary or acquired bleeding diathesis, platelet transfusion is indicated if platelet count  $<50,000/\mu\text{L}$ . FFP transfusion is indicated for patients on warfarin with an elevated INR and significant bleeding, liver failure, or massive transfusion ( $>10$  units PRBC in 24 hours). PCC is used for warfarin reversal (FFP, if not available). In a massive transfusion, plasma, platelets, and red blood cells should be given in 1:1:1 ratio.

### Inotropes

If volume resuscitation does not improve the patient's hemodynamic status and MAP remains below 65, inotropes may be used. Inotropes are also used in cardiogenic shock for depressed LV function. Norepinephrine (2-10 mcg/min) stimulates alpha and beta-adrenergic receptors, increasing peripheral vascular tone. Dobutamine (2.5-15 mcg/kg/min) may improve myocardial contractility and augment diastolic coronary blood flow

through beta-1 adrenergic agonist effect, and it may cause mild peripheral vasodilation through beta-2 adrenergic agonist effect. Dopamine at moderate doses (5-10  $\mu\text{g/kg/min}$ ) has alpha and beta-1 adrenergic effects.

### Treatment Success

Monitoring fluid status is encouraged by using a urinary catheter, intra-arterial blood pressure measurements, and central venous pressure monitoring.

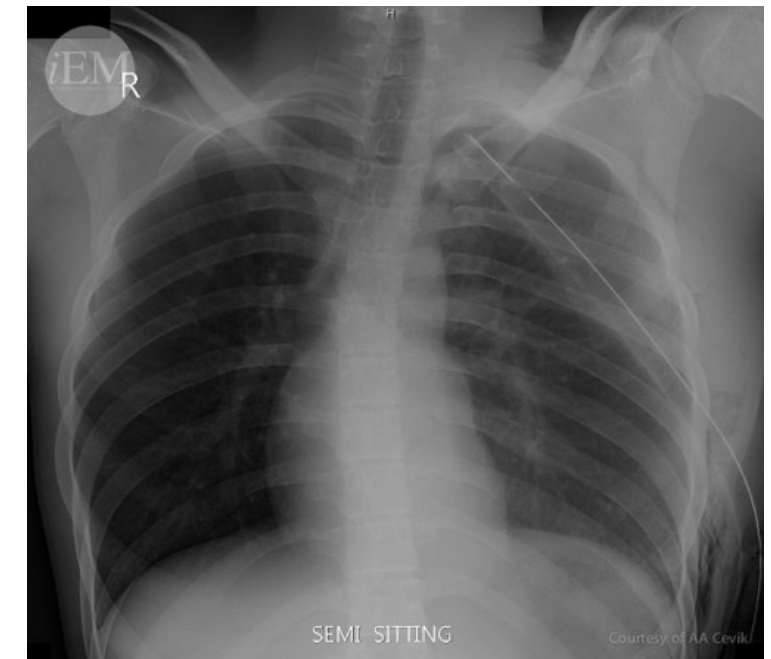
When patients' hemodynamic status become normal (blood pressure, heart rate and urine output) and necessary volume restored. These help to maximize tissue oxygenation, resolution of acidosis and decrease lactate levels. These are the findings of successful resuscitation.

### Medications

- Distributive shock
- The treatment depends on the specific cause of shock.



**Image 3.25** Chest X-ray shows position of the chest tube in a patient with pneumothorax.



An intra-aortic balloon pump decreases afterload and increases diastolic BP and bridge to revascularization or valvular repair. Also, cardiogenic shock may necessitate emergent angiography or surgical procedures such as bypass or valve repair.

## Disposition Decisions

Despite proper treatment, the mortality rates from severe shock can exceed 50 percent. Even after aggressive treatment in the ED, ICU admission is required.

- In septic shock, it is important to start early broad-spectrum antibiotics:
  - Neonates: Ampicillin (150-200 mg/kg) with Cefotaxime (150-200 mg/kg)
  - Children: Vancomycin (15-20 mg/kg) and Cefotaxime (150-200 mg/kg)
  - Adult: Vancomycin (15-20 mg/kg) with Piperacillin-Tazobactam (80-100 mg/kg)
  - Consider low-dose corticosteroids to treat relative adrenal suppression if refractory hypotension is present despite fluids and inotropes.
- Anaphylactic shock
  - Epinephrine is the first line of treatment.
    - Adults: 0.3-0.5 mg of 1:1000 solution IM q5-10 minutes
  - Pediatrics: 0.01 mg/kg, max 0.3 mg of 1:1000 solution IM q5-10 minutes
  - Histamine blockers.
    - Diphenhydramine (H1 blocker): (25-50 mg IM/IV, pediatric 1 mg/kg IM/IV)
    - Cimetidine or ranitidine (H2 blockers).
  - Aerosolized albuterol and ipratropium are important in anaphylactic shock.
  - Steroids should be considered

## Procedures

In the management of obstructive shock, it is essential to remove obstruction by pericardiocentesis in cardiac tamponade tamponade, by needle thoracocentesis followed by tube thoracostomy in tension pneumothorax, by thrombolysis in massive P.E., and afterload reduction until definitive treatment in aortic dissection.



References and Further Reading, click [here](#)

## Chapter 4

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# Selected Cardiovascular Emergencies



# Abdominal Aortic Aneurysm (AAA)

by Lit Sin Quek

## Case Presentation

*A 75-year-old obese man comes to the emergency department. He has history C.O.P.D., hypertension. He is a smoker and on regular follow-up with primary care. He describes sudden onset severe flank and back pain for past 2 hours. He denies any chest pain or dyspnea. He informs the physician about his chronic abdominal pain. His initial vital signs are HR 98 bpm, RR 24/min, BP 190/105 mmHg, T 36.9C. His examination revealed mild **abdominal pain** without rigidity or rebound tenderness. Bedside ultrasonography performed and the result is shown below.*

Image 4.1



Audio is available [here](#)



## Introduction

Abdominal Aortic Aneurism (AAA) rupture is one of the serious problems which should be suspected in every hypotensive elderly with abdominal pain. This chapter's learning objectives are;

- Understand the epidemiology and pathogenesis of AAA
- Appropriate diagnostic measures
- Clinical Key Points
- Recognize indications for referral

## Abdominal Aortic Aneurysm (AAA)

AAA is defined by International Society for Cardiovascular Surgery and Society for Vascular Surgery as “a focal dilation (widening) of the abdominal aorta where the diameter is at least 50% larger than the expected normal diameter for that individual.” However, most clinicians will consider the diagnosis of an AAA if the diameter greater than 3 cm.

### The leading causes of AAA are;

- Atherosclerosis
- Genetic predisposition (weakening vs. occlusion)
- Connective tissue diseases
- Marfan's, Ehlers-Danlos
- Infection (Syphilis, salmonella, others)

### Epidemiology

There are many factors affecting AAA development. These factors are very critical to reaching the diagnosis and knowing those can help you during the history taking, even in the management of the patient. Factors Associated with AAA are as follows;

- Old age: generally above 55 considered is a risky cut-off for AAA.
- Gender: Men develop AAA 4-5 times more often than women
- Ethnicity: White people develop AAA more frequently than other ethnicities

- Cardiovascular risk factors and vascular bed affection: People with coronary artery disease and peripheral artery disease are more prone to have AAA.
- Family history: A family history of AAA increases the risk of developing AAA. The risk of developing an AAA may reach 20% among brothers of a patient with a known AAA.
- Smoking: It is a risk factor for many diseases. Number of years of smoking is related to high risk.
- Diabetes mellitus: There is a negative association with diabetes mellitus and AAA.
- Hypertension: It is a poor predictor for AAA development but important risk factor for expansion and rupture.
- Lipid: There is no and weak correlation between risk for AAA and high serum triglyceride and cholesterol, respectively.



- Emphysema: It is the strongest independent risk factor for rupture. Prevalence is 5% to 7% of people over the age of 65 in the United States. There is a 3:1 ratio of men to women. After age 65, the prevalence of 3 cm aneurysms in men increases by approximately 6% per decade.

### Types of AAA

- Saccular aneurysm – is an outpouching arising from one part of the aorta, has a neck, and does not involve the entire circumference of the aorta.
- Fusiform aneurysm – is tubular in shape, involves the entire circumference of the localized aorta, and has no neck.
- Pseudoaneurysm – dilatation is only at the outside layer of the aorta (tunica adventitia)
- Mycotic aneurysm – a rare aneurysm caused by a fungal infection which may be associated with immunodeficiency, IV drug abuse, heart valve surgery.

### Presentation

Abdominal/back pain, a pulsatile mass, and hypotension are known as the classic triad, but only seen 1/3 of the patients. So, you have to lower your threshold to be suspicious for cases showing epidemiologic warnings described above.

### Critical Bedside Actions and General Approach

As described in many other chapters (e.g., Shock), the primary goal is the resuscitate any unstable patient. Therefore, airway, breathing, circulation should be evaluated immediately and resuscitative measures implemented. If the rupture is suspected, immediate surgical consultation and blood transfusion to the patient is a must. Do not delay the definitive treatment which is surgery.

### Differential Diagnoses

- Renal colic
- Diverticulitis
- GI bleeds

• Myocardial infarction

- Musculoskeletal back pain

The patients may have a variety of differential depending on their symptoms. The important clue to keep in mind is each of these specific diseases shows their specific symptoms, and as a rule of thumb, these symptoms may be indirectly mimicking AAA, especially elderly patients and patient who have risk factors.

### History and Physical Examination Hints

Many of the patients are elderly. Because of their pain sensation affected by multiple comorbidities, AAA patients may not give clear history hints to physicians. Most of the times, symptoms are very subtle unless hypotension and shock situation in rupture. The patients showing epidemiologic risk factors should be questioned very carefully.

Physical examination of the patients should include relevant organ systems

that patient having risk factors or symptoms. Specific attention should be given to understand instability.

Specific exam for AAA includes deep gentle palpation, above the umbilicus, left of midline, continuous over several heartbeats. Bleeding into retroperitoneum may create doughy abdomen. Hypotension also minimizes pulsations.

Some facts;

- 38% patients AAA initially detected by physical examination
- 62% found incidentally on imaging studies done for other indications
- AAA detected by physical examination had lower BMI but there was no difference in AAA size
- 43 % of AAA detected on radiologic examination is palpable and should have been detected on physical examination.
- 23% AAA were not palpable on pre-operative physical examination, even when the diagnosis was known.
- Obese patients had only 15% of AAA detected by physical examination, and only 33% were palpable.

## Emergency Diagnostic Tests and Interpretation

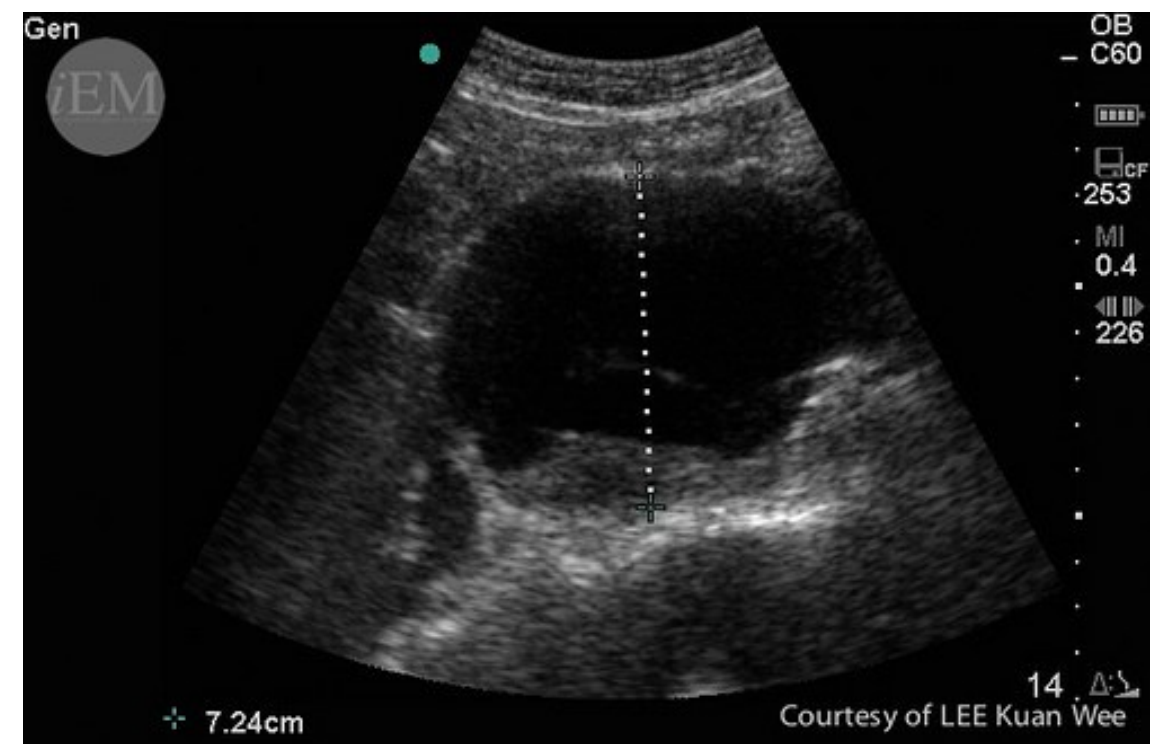
### Laboratory tests

The most important issue for these patients is bleeding. Therefore, type and cross-match blood is the most critical test. CBC, Urea/Creatinine, coagulation studies and urinalysis are other tests.

### Imaging modalities

- Ultrasound provides low cost, reliable, fast and safe approach. However, it is operator dependent modality. Poor imaging above renal vessels, obesity, intestinal gas, or very painful abdomen may affect the proper investigation. Please see RUSH protocol chapter to learn more about aortic ultrasonographic evaluation.

Image 4.2 US - AAA



Abdominal aorta investigation with ultrasound. Tutorial in 3 minutes take a look this [video](#).

- CT Scan with contrast is a gold standard (Image 4.3). It shows better demonstration extent and complications of an aneurysm, retroperitoneal blood because of rupture, and dissection. However, the patient instability affects the usage of this imaging modality.

**Image 4.3** CT scan - AAA



- MRI has no advantage over CT scan.
- The angiogram is not preferred for diagnosis but good for pre-op “mapping.”

- Abdominal X-Ray/KUB may incidentally show findings of AAA. AAA can be seen in 60-75% of cases in the x-ray with the calcification of aortic wall or paravertebral mass.
- Cross-table lateral most helpful view and a negative study is not helpful.

## Emergency Treatment Options

### Medications

There are no specific medications for AAA patients. However, some patients may require blood pressure and arrhythmia management. In the unstable patients, intubation with rapid sequence intubation (RSI) protocol, fluid and blood replacement should be considered. Analgesics also an important part of the treatment.

### Procedures

Any critically ill patient who diagnosed AAA (potentially rupture) should immediately be intubated and airway secured. This also prepares the patient for the operation theatre. Some patients may have no peripheral IV access because of their shock situation. These patients require an intraosseous line or central I.V placement. Although these resuscitative measures keep the patient alive and any ruptured patient should directly go to the operation theatre, you should also know some other red flags for the indications for repair of AAA.

- Size more than 5.5 cm. However, 5.0 cm still used in common practice by many surgeons.
- Symptoms such as abdominal or back pain, to groin in some cases or tenderness of AAA.
- Risk of Rupture: Emphysema, smoking, hypertension increase likelihood of rupture. Regarding Powell et al.'s study aneurysms less than 5.5 cm in diameter has less than 1% of rupture in one year. Above 5.5 cm risk is between 9.4% to 32.4 (more than 7 cm).
- Rupture ("Leak") as we discussed above.

Other treatment options and management strategies should be thought in stable AAA patients such as;

- Observation: Small aneurysms < 5 cm
- Elective repair: Open surgical repair or endovascular (stent-graft) repair

Additional Information: see the video on repairs – [link](#)

## Clinical Key Points – putting it all together

- Abdominal aortic aneurysms are asymptomatic until they rupture, resulting in a mortality of 85 to 90%.
- Urgent repair is the only definitive option for symptomatic patients.
- Although the optimal group to be screened remains controversial,
- Smoking men or women 65 to 75 years of age and 65 to 75 years of age non-smoking man should undergo screening and selective screening, respectively.
- The threshold for elective repair is an aortic diameter of 5.5 cm in men and 5.0 cm in women, but this may vary with practices.
- Endovascular repair results in lower perioperative morbidity and mortality than open repair, but the two methods

are associated with similar mortality up to 10 years.

- Patients treated with endovascular repair require long-term surveillance owing to a small risk of aneurysm sac reperfusion and late rupture.
- Decisions regarding prophylactic repair — whether to pursue and what type of repair to perform must take into account anatomy (not all situations can undergo endovascular repair), operative risk, and patient preference.

## Disposition Decisions

### Admission criteria

- All unstable patients should be transferred to operation theatre immediately.
- Stable patients with high risk of rupture, if they are not going to operation theatre, they can be admitted into ward or ICU depending on institution protocols.

## Discharge criteria

- Asymptomatic patients only patient group can be discharged if they do not have any risk factor for rupture. Patient with risk factors should be evaluated carefully. If they are decided to discharge, close follow-up in the clinic should be arranged. The outpatientclinic folow-up for other patients must also be arranged before their discharge from the emergency department. Instruction specific to AAA should be given to patients.

**References and Further Reading**, click [here](#)



# Acute Coronary Syndrome (ACS)

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by Khalid Mohammed Ali, Shirley Ooi

## Case Presentation

*A 46 years old man with a past medical history of hypertension and hyperlipidemia developed central crushing chest pain associated with sweating and shortness of breath while driving. He presented to the emergency department 1 hour after the onset of chest pain. On physical examination, his vital signs were as follows; pulse rate: 60 beats/min, blood pressure: 100/50 mm Hg, respiratory rate: 20/min, SpO2 98% on room air. Patient has no leg edema, new murmur or features of heart failure.*

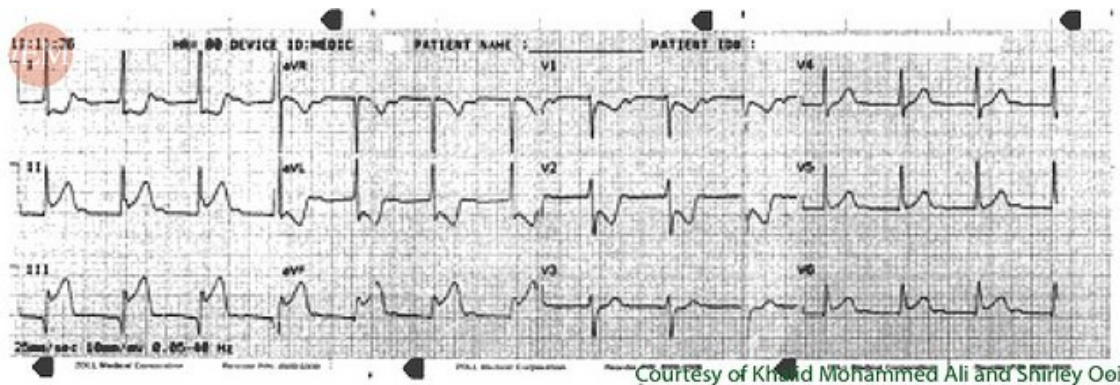
*His ECG is as is given.*

*What are the ECG features? What is the diagnosis?*



Audio is available [here](#)

**Image 4.4**



Courtesy of Khalid Mohammed Ali and Shirley Ooi

## Answer

ECG features are ST elevation more than 2 mm in inferior leads (II, III, AVF) with reciprocal changes (deep ST depression in lateral leads of I, AVL) and ST depression in lead V1, V2 which indicate the involvement of posterior wall. The ECG is diagnostic of inferior and posterior wall ST elevation MI.

## Definition

Acute coronary syndromes (ACS) include conditions that share the same pathophysiology of myocardial ischaemic states, i.e., unstable angina (UA), non-ST elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

## History and Physical Examination Hints

The patient may experience the following symptoms:

- Chest pain described as pressure, squeezing or a burning sensation across the precordium and may radiate to the neck, shoulder, jaw, back, upper abdomen, or either or both arms.
- Shortness of breath especially exertional dyspnoea
- Palpitation
- Diaphoresis from sympathetic stimulation
- Nausea from vagal stimulation
- Decreased effort tolerance

It is important to ascertain the onset of chest pain as it will affect the management of STEMI. Other important questions to ask are the chest pain character, radiation, associated symptoms, relieving and exacerbating factors, especially exertion.

Do not forget to ask about the risk factors. You can use the TIMI score.

**TIMI** (Thrombolysis in Myocardial Infarction) investigators have developed a 7-variable risk stratification tool that predicts the risk of death, re-infarction, or urgent revascularization at 14 days after the presentation:

- $\geq 65$  years of age
- Presence of  $\geq$  three cardiac risk factors

- Prior coronary artery stenosis of  $\geq 50\%$
- $\geq$  two angina events in the preceding 24 hours
- Aspirin use in the previous seven days
- ST-segment deviations of  $\geq 0.5$  mm on ECG at presentation
- Positive cardiac biomarkers

Patients are considered to be high risk if their TIMI risk score is  $\geq 5$  and low risk if the score is  $\leq 2$ . High-risk patients have a more significant benefit from early percutaneous coronary intervention and use of adenosine phosphate inhibitor and low molecular weight heparin than lower risk patients.

Most of the cases with ACS have a normal cardiovascular examination. In a busy emergency department where time is of an essence, targeted physical examination in the patient with ACS is important to rule out complications and possible differential diagnosis.

The following are essential components:

- Differential pulse and BP between both arms, which if present may indicate the possibility of aortic dissection
- Tachypnoea, pitting leg edema and raised jugular venous pressure with crackles in the base of the lung or only crackles in

the base of the lung may indicate either existing heart failure or an acute one secondary to acute myocardial ischemia

- A new cardiac murmur may indicate acute valvular insufficiency or rupture interventricular septum
- Distant heart sound on auscultation of precordium may indicate acute pericardial effusion secondary to rupture of a free ventricular wall or acute aortic dissection with extension to precordium.

In addition to the above, it is essential to check the vital signs carefully. If the patient has hypotension with acute myocardial ischemia, this may indicate cardiogenic shock. Tachycardia may range from sinus tachycardia to ventricular tachycardia; bradycardia, on the other hand, may range from sinus bradycardia to third-degree heart block.

## Differential Diagnosis

There are many critical differential diagnoses when we consider A.C.S. Please look for all in the given table. However, Unstable angina, Acute myocardial infarction, Acute pulmonary embolism, Acute aortic dissection, Tension pneumothorax, Oesophageal rupture (Boerhaave's syndrome) are the life-threatening ones. We advise you to read these chapters from multiple resources to feel confident.

See the following table:

**Table 4.1** ACS/Chest Pain Differential Diagnosis

LIFE THREATENING	CVS	RESPIRATORY	GI	REFERRED PAIN
Unstable angina	Stable angina	Pneumonia	Gastro-oesophageal reflux	Sub phrenic abscess / inflammation
Acute myocardial infarction	Acute pericarditis	Simple pneumothorax	Oesophageal spasm	Hepatobiliary disease
Acute pulmonary embolism	Myocarditis			
Acute aortic dissection				
Tension pneumothorax				
Oesophageal rupture (Boerhaave's syndrome)				

## Acute Complications

The acute complication which we may see in the emergency department includes the following:

- Acute pulmonary edema due to acute myocardial ischemia which leads to decrease effective ejection fraction and heart failure
- Mechanical complications include rupture of papillary muscles, free left ventricular wall, and interventricular septum

- Arrhythmias include tachyarrhythmias and bradyarrhythmias
- Cardiogenic shock
- Cardiac arrest in the form of ventricular fibrillation

## Decision Making Process and Reaching The Diagnosis

There are three pillars of diagnosis: history, ECG, and cardiac enzymes.

### Clinical features of unstable angina include the following:

- Unstable angina differs from stable angina in that the chest pain is usually more intense, easily provoked, more prolonged, more frequent and more severe. All first presentation of angina should be regarded as unstable. In unstable angina typically there is either no ECG changes or non-specific ECG changes, the patient is usually chest pain-free on presentation to the emergency department, and the cardiac enzymes will be normal.
- NSTEMI should be diagnosed in any patient whose cardiac enzymes are raised without evidence of ST elevation MI. An NSTEMI does not need to have ECG changes at the time of presentation. The ECG may show the following:

1. ST-segment depression

2. The transient ST-segment elevation that resolves spontaneously or after glyceryl trinitrate treatment
3. T-wave inversion
4. Evidence of previous myocardial infarction
5. Left bundle branch block
6. Minor non-specific changes

The ECG can also be normal. It should not show persistent acute ST-segment elevation.

- STEMI (ST-segment elevation MI) is a true cardiac emergency. The criteria of diagnosing ST-segment elevation MI on ECG are: New ST elevation at the J point in at least two contiguous leads of  $\geq 2$  mm in leads V2–V3 and/or of  $\geq 1$  mm in other contiguous chest leads or the limb leads.

**Table 4.2** ST Segment Changes And Its Anatomical Relation In Acute Myocardial Infarction

LOCATION	LEADS	ST SEGMENT CHANGES
Anterior wall	V1 -V4	Elevation
Inferior wall	II, III, aVF	Elevation
Posterior wall	V8 and V9 V1-V3	Elevation Depression
Right Ventricular wall MI	V4R, V5R, V6R	Elevation
Lateral wall	I, aVL, V5 and V6	Elevation



There are other causes of ST elevation should be known to differentiate it from the ST elevation of myocardial infarction. These are;

- Acute pericarditis
- Benign early repolarization
- Brugada's Syndrome
- Hyperkalemia
- Left Bundle Branch Block
- Left ventricular aneurysm
- Left ventricular hypertrophy
- Normal variant
- Osborn wave of hypothermia
- Prinzmetal's angina
- Ventricular paced rhythm

In general, the difference between unstable angina and NSTEMI/STEMI is an absence of cardiac enzymes abnormalities.

Cardiac enzymes (CKMB, Troponin T or I) are highly sensitive to cardiac muscle injury. Another lab investigation is full blood count, urea, and electrolyte. A chest x-ray may give clues to

acute pulmonary congestion or indicate the diagnosis of other conditions like pneumothorax or acute aortic dissection.

## Emergency Treatment Options

### Initial Stabilization

In typical emergency medicine room, once a patient presents with chest pain suspecting of acute myocardial ischemia should be seen in the monitored area, the patient should remain under continuous cardiac monitoring, HR, BP, and SpO2.

The proper approach will consist of all following:

- 📍 ECG should be done immediately or within the first 10 minutes by the emergency room staff nurse, which should be interpreted by a senior doctor.
- 📍 Targeted history of onset of chest pain, associated symptoms and risk factors.
- 📍 Targeted examination to exclude potential differential diagnosis and complications of acute myocardial ischemia.
- 📍 IV cannula will be set, and blood will be sent for full blood count, urea and electrolytes, cardiac enzymes (CKMB, troponin I or T).
- 📍 Radiological examination of the chest will be required only to diagnosis acute pulmonary edema, rule out possible differentials like pneumothorax or aortic dissection.

- 📌 Antiplatelet typically aspirin 300 mg and either Ticagrelor 180 mg or Prasugrel 60 mg.
- 📌 GTN sublingually or spray to relieve chest pain, if chest pain persists after two sublingual GTN tablet, proceed with GTN infusion especially if the patient has concomitant hypertension or heart failure.
- 📌 IV morphine with anti-emetic if chest pain persisted and titrated according to the response of the patient.
- 📌 Oral beta blocker if no contraindication within 24 hours.
- 📌 Definitive treatment depends on which condition within the acute coronary syndrome is diagnosed.
- 📌 STEMI should undergo reperfusion therapy preferably percutaneous coronary intervention (PCI) or intravenous thrombolytic therapy
- 📌 Unstable angina and NSTEMI: medical therapy of antiplatelet, anticoagulant and beta blocker followed by admission to hospital and arranging of urgent PCI
- 📌 For STEMI, time is muscle, the sooner the PCI or thrombolysis, the better prognosis is.

**Table 4.3** Advantages And Disadvantages Of Thrombolysis Versus PCI

	THROMBOLYSIS	PCI
Advantages	Rapid administration Widely available Convenient	Better clinical efficacy i.e. superior vessel patency, TIMI grade 3 flow rates and reduced occlusion rates Less haemorrhage Early definition of coronary anatomy allows tailored therapy and more efficient risk stratification
Disadvantages	Patency ceiling, i.e. infarct-related artery is restored in only 60-85% of patients, with a normal TIMI grade 3 epicardial coronary flow in only 45-60% of patients  Less clinical efficacy, i.e. optimal reperfusion is not achieved in more than 50% of patients, and re-occlusion of infarct vessel occurs in 5-15% of patients at week 1 and 20-30% within 3 months  Risk of haemorrhage	Delay limits efficacy Less widely available Requires expertise

In the absence of PCI, thrombolysis is alternative, and the following should present for the patient to be a candidate of thrombolysis:

- Typical chest pain of AMI
- ST-segment elevation fulfilling the criteria stated above
- Chest pain <12 hours from onset
- Patients <75 years of age

**Table 4.4** Contraindications Of Intravenous Thrombolysis

ABSOLUTE	RELATIVE
History of intracranial haemorrhage History of ischaemic stroke in the past 3 months (except acute ischaemic stroke within 3 hours) Presence of cerebral vascular malformation or intracranial malignancy Suspected aortic dissection Bleeding diathesis or active bleeding (except menses) Significant head trauma or facial trauma in the past 3 months	Severe hypertension (blood pressure > 180/ 110 mm Hg) History of ischaemic stroke >3 months Presence of dementia Known intracranial disease that is not an absolute contraindication Traumatic or prolonged cardiopulmonary resuscitation (CPR) that lasted more than 10 minutes Major surgery within 3 weeks Presence of active peptic ulcer Internal bleeding within the last 2 to 4 weeks Non-compressible vascular punctures. Pregnancy On warfarin therapy For streptokinase, prior exposure (more than 5 days ago) or history of allergic reaction

In the acute management of acute coronary syndromes, please take note of the following:

If the patient is in shock, always look for precipitating causes:

1. Do a gentle rectal examination to look for gastrointestinal bleeding.
2. Is the patient bradycardic? Treat according to ACLS guidelines.
3. Is the patient tachycardic? Treat according to ACLS guidelines.
4. Does the patient have a right ventricular infarct?
  - a. Do right-sided leads in the presence of ST elevation in II and III and aVF as in inferior AMI (Look for at least 1 mm ST elevation in V4R, V5R, and V6R.
  - b. If so, give the fluid challenge of 100-200 ml normal saline over 5 to 10 minutes and assess response.
  - c. This can be repeated if the patient does not become breathless and there are no clinical signs of pulmonary edema.
  - d. Start inotropes (IV dobutamine/dopamine 5-20 µg/kg/min) if the blood pressure remains low despite IV fluid administration of 500 ml.

5. Is the patient in cardiogenic shock because of mechanical complications, e.g., papillary muscle dysfunction or rupture, septal rupture or cardiac tamponade from free wall rupture?
- a. Call the cardiologist and cardiothoracic surgeon.
  - b. Meanwhile, start inotropic support, e.g. IV dobutamine/ dopamine 5-20  $\mu\text{g}/\text{kg}/\text{min}$
  - c. Catheterize the patient to measure the urine output.
  - d. Admit to the coronary care unit or send the patient to the cath lab.

**References and Further Reading**, click [here](#)

# Acute Heart Failure (AHF)

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by Walid Hammad

## Case Presentation

*An ambulance crew rushes into your emergency department (ED) with a 56-year-old man. He is severely short of breath, sitting upright on the stretcher, using his accessory respiratory muscles, and gasping for air. You find that he is diaphoretic, tachypneic, and in severe respiratory distress. You ask him, “What’s going on?” He replies: “I...can’t...(pauses and inhales a shallow breath)...breathe!”*

*The paramedics inform you that they received a call from the patient’s wife about 6:30 that morning, saying that her husband was short of breath and sweaty and that he had vomited once. The wife told them that she and her husband had returned from a long trip the night before and that her husband had not taken his “water pills” because he did not want to stop for frequent restrooms breaks during their drive. When they got home, he still did not take his pills because he*



Audio is available [here](#)



*wanted to sleep through the night. His breathing problems woke him during the night, and he tried to get more comfortable by adding pillows under his head to the point that he was almost sitting up in bed.*

*You thank the paramedics and turn back to the patient, who now looks even worse. He is more short of breath, and you sense that he is getting tired, about to give up. He looks like he is about to collapse. What is your next step?*

## **Critical Bedside Actions and General Approach**

The first step in managing such a patient, as for most ED patients, is measuring vital signs. This information will help you identify a part of the pathology. For example, if the patient is hypertensive, he could be in acute heart failure; on the other hand, if the patient is hypotensive, he could be in shock. Similarly, if the patient is tachycardic, his symptoms could be caused by the very fast heart rate; conversely, if he is bradycardic, he could have symptomatic bradycardia. The vital signs will guide your treatment options. When you examine the patient described in this case report, you find his blood pressure (BP) to be 265/145 mm Hg, his heart rate (HR) to be 138 beats/min, his respiratory rate at almost 40 breaths/min, and his pulse oximetry reading is 92% on 4 liters of oxygen delivered by nasal cannula. (Note: A patient who is severely short of breath might be breathing through his or her mouth, so a nasal cannula may not be of great benefit; in these patients, the use of a face mask might be prudent.)

This patient's clinical presentation and vital signs represent a clinical pattern of acute heart failure (AHF) with severe acute pulmonary edema (APE) secondary to acute left ventricular failure (LVF). The LVF and subsequent pulmonary edema are secondary to any or a combination of (1) an increased preload, (2) a decreased left ventricular ejection fraction, or (3) an extremely elevated blood pressure.

On the other hand, a patient who presents with the same clinical picture but with a low BP instead of a high one could have APE or acute LVF secondary to cardiogenic shock. In this scenario, your treatment choices will change, and your strategy will be directly opposite that for a patient with elevated BP. It is crucial to make this distinction early because the administration of vasodilators in high doses to a patient in cardiogenic shock could have a devastating outcome.

The second step in management is the clinical examination. In a patient with AHF with consequential severe APE, the clinical picture might resemble severe Stage D congestive heart failure (CHF), but with a swifter, more acute onset. Generally, the patient is in moderate to severe distress, is uncomfortable, and is usually diaphoretic, with jugular venous distention (JVD) and bilateral rales on lung examination. Depending on how long the patient has been in severe CHF, the rales might be basal initially but then heard in all lung fields, up to the apices, in late stages or even audible without a stethoscope.

Hepatic engorgement, a positive abdominojugular test, and bilateral lower extremity edema are signs of chronic CHF and might not have developed yet in a patient with acute left heart failure. These signs start to manifest when the pulmonary arterial wedge pressure increases. Think about the pathology: the heart (the pump or engine), specifically the left ventricle, is unable to pump the blood against the overwhelming resistance generated

by the increased systemic vascular resistance (SVR) that is generating an extremely high BP in the aorta. With such elevated BP, the left ventricle is unable to offload the suitable amount of blood with each stroke, i.e., stroke volume (SV) is decreased, leading to a decrease in cardiac output (CO). With time, if the preload does not decrease and the afterload continues to increase, the blood will back up behind the left ventricles (in the lungs), causing the lungs to become engorged with accumulated blood and thus increasing transudation of protein-poor fluid into the interstitial space and the alveoli. This cascade compromises the air exchange mechanisms, causing the patient to manifest signs and symptoms of respiratory failure, presenting clinically as tachypnea and hypoxia.

## The First 5 to 10 Minutes

The main pathology in AHF is the extremely high SVR in the heart, so the treatment modalities should focus on decreasing the resistance, i.e., decreasing the blood pressure (afterload) or decreasing preload. The mean arterial pressure (MAP) can be used as a treatment guideline. It is determined by the cardiac output (CO) and SVR:  $MAP = CO \times SVR$ . Note that about 50% of patients presenting with APE are euvolemic rather than hypervolemic and that the treatment options should focus on volume redistribution rather than volume removal.

Since you do not want to decrease CO in a patient who is barely perfusing because of the elevated BP, your best bet is to work on

decreasing preload and/or SVR and thus decrease the MAP. This can be achieved by several means. However, in this scenario, in which the patient is extremely ill and needs the MAP to be dropped quickly, we head directly to the rapid-onset options—nitro derivatives (fast-acting nitroglycerin derivatives) and BIPAP or CPAP.

## Differential Diagnoses

- Acute Kidney Injury
- Acute Respiratory Distress Syndrome
- Bacterial Pneumonia
- Cardiogenic Pulmonary Edema (secondary to cardiogenic shock)
- Chronic Obstructive Pulmonary Disease (COPD)
- Cirrhosis of the liver
- Community-Acquired Pneumonia
- Emphysema
- Goodpasture Syndrome
- Idiopathic Pulmonary Fibrosis
- Interstitial (nonidiopathic) Pulmonary Fibrosis

- Myocardial Infarction
- Nephrotic Syndrome
- Neurogenic Pulmonary Edema
- Pneumothorax
- Pulmonary Embolism
- Respiratory Failure
- Venous Insufficiency
- Viral Pneumonia
- Others

## History and Physical Examination Hints

- JVD might be present, indicating increased central venous pressure (CVP) resulting from pulmonary edema and increased right ventricular pressures.
- An extremely elevated BP in a previously relatively healthy patient presenting with APE points to acute heart failure.
- Bilateral pulmonary rales are typical of APE but not specific. Rales on only one side could suggest other causes such as pneumonia or emphysema (dry crackles).

- APE can also present as bilateral wheezing (cardiac asthma); however, this presentation should not be confused with the wheezing associated with pure reactive airway disease or asthma.
- If the patient presents with altered mental status or has an abnormal neurologic exam, the APE might be neurogenic pulmonary edema.
- If the patient is receiving dialysis or has nephrotic syndrome, cirrhosis, or other causes of volume overload, the backbone of treatment will be diuresis rather than redistribution (preload or afterload reduction).
- If lung sounds are unequal, the patient might have a spontaneous pneumothorax. If the patient is as sick as the one in our scenario, he or she could be experiencing cardiac tamponade (pay attention to the position of the trachea).

## Emergency Diagnostic Tests and Interpretation

### Bedside Tests

1. By placing the head of the stretcher at a 45-degree angle, you should be able to assess the patient for JVD.
2. An electrocardiogram (ECG) might show sinus tachycardia, atrial fibrillation, or another arrhythmia (tachycardia or bradycardia), suggesting the reason for a decrease in cardiac

output ( $CO = HR \times SV$ ). The ECG could also reveal a left ventricular strain pattern.

3. With proper training, emergency physicians can reliably obtain the following information with an ultrasound examination performed at the bedside:
  - Determine left ventricular ejection fraction as a broad categorization (normal, moderately reduced, severely reduced) – [link](#)
  - Check for pulmonary congestion/edema – [link](#)
  - Evaluate volume status by examining the inferior vena cava – [link](#)

### Laboratory Tests

1. Complete Blood Count (CBC): May show an elevated white blood cell (WBC) count, which may indicate an infectious cause rather than a cardiac cause. However, stress in itself can induce hypoxia and shortness of breath, which can cause margination of WBCs, leading to an elevated WBC count. A patient with severe chronic obstructive pulmonary disease (COPD) or Goodpasture syndrome is likely to be taking corticosteroids, which can raise the WBC count.
2. Comprehensive Metabolic Panel (CMP): May indicate renal failure if the SVR is so high that it is causing severe spasm in the renal artery and thus impairing kidney function, especially if

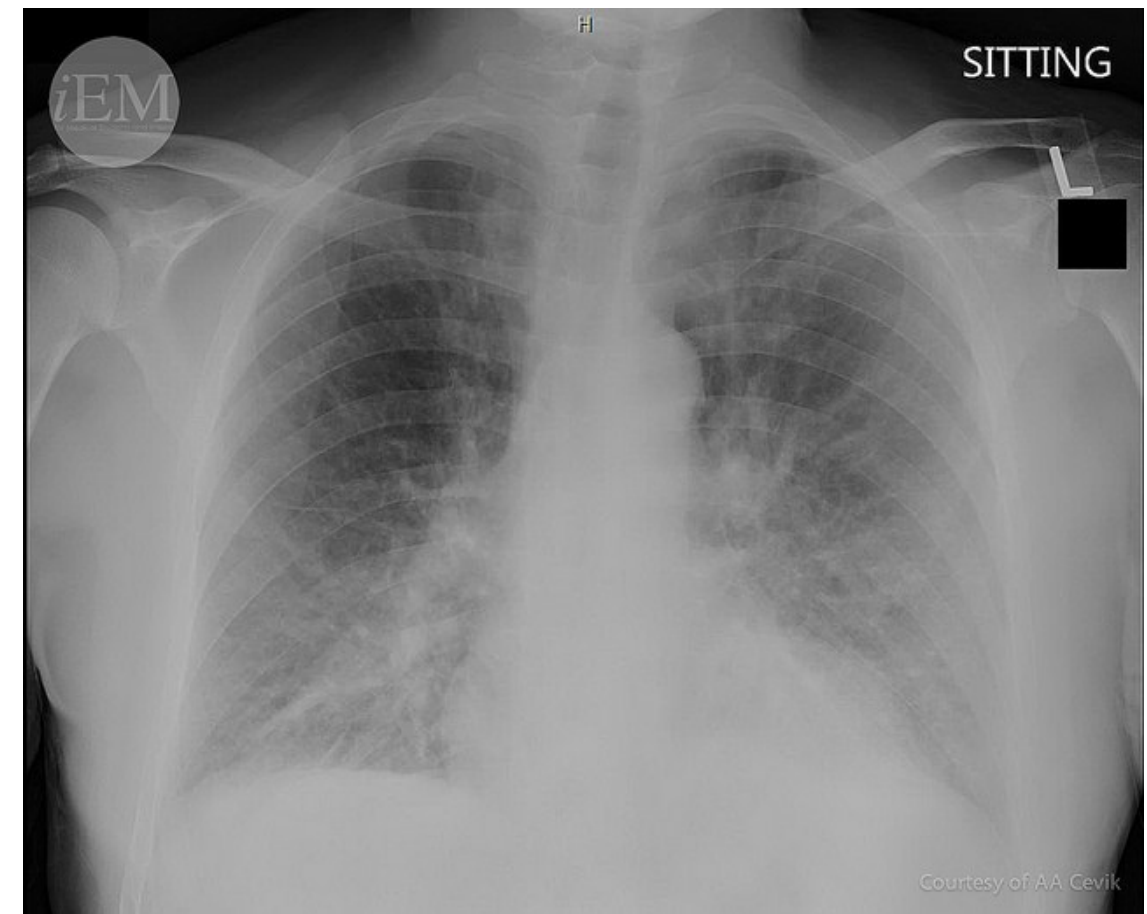
the patient has other comorbidities that predispose him/her to kidney injury. The patient might be alkalotic in response to tachypnea, which presents as low CO<sub>2</sub>. He may also present with acidosis due to elevated lactate levels resulting from tissue hypoxia. Liver function test results could be elevated, especially if the patient has long-standing CHF that is causing hepatic engorgement.

3. Pro-BNP: The b-type natriuretic protein (BNP) concentration will be elevated in a patient with acute heart failure, but usually the values are not very high. Be careful: pro-BNP could also be elevated in patients with large pulmonary embolisms that are causing right ventricular strain. Cardiac enzymes might be slightly elevated due to the myocardial strain; significantly elevated numbers warrant consideration of Acute MI.

## Imaging Modalities

A chest x-ray (obviously a portable frontal view in our patient) would show pulmonary congestion, with cephalization of the pulmonary vessels, Kerley B lines, peribronchial cuffing with air bronchograms, a “bat wing” pattern, and possibly, though not always, an increase in cardiac shadow size. Note that not all these findings may be present on the chest x-ray.

Image 4.5



## Emergency Treatment Options

### Initial Stabilization

1. If the patient is lying down flat, move him/her into a sitting position, which should lessen the pooling of blood in the lungs and allow the utilization of the superior lung fields for aeration and gas exchange.
2. Place the patient on supplemental oxygen.
3. Obtain intravenous access quickly.



## Non-invasive positive pressure ventilation (NIPPV)

NIPPV for cardiogenic pulmonary edema is an effective and safe modality in adult patients with APE. There is a potential benefit of NIPPV in reducing mortality.

## Medications

- Nitro derivatives: Nitrogen inhibits the motor function of the smooth muscles in the systemic vasculature, leading to vasodilation and a decrease in SVR.

- Nitroglycerin: Reduces preload.

- Nitroglycerin has been found to be safe for use in patients with acute heart failure and improves short-term outcomes in EDs and ED-like settings.
- Nitroglycerin is a rapid-onset, short-acting smooth muscle relaxant that reduces preload through venous dilation and, in high doses, reduces afterload through arteriolar dilation.
- Sublingual tablets dissolve under the tongue and are manufactured in 400-mcg minitables. After you have confirmed the diagnosis by exam and the monitor is indicating a high blood pressure, place one, two, or even more tablets under the patient's tongue (up to one tablet per minute).

- Nitroglycerin might cause hypotension, which is usually temporary and associated with overall clinical improvement. However, the persistence of the hypotension after the nitroglycerin is stopped might indicate a right ventricular malfunction, e.g., right ventricular MI or volume depletion.

- Intravenous nitroglycerin can be administered, starting as a drip at 0.5-0.7 mcg/kg/min and then increased by 10-20 mcg/min q3-5 minutes up to 200 mcg/min. The BP must be monitored closely during administration.

- Transdermal nitroglycerin (1-5 cm) can be applied to the chest wall.

- Pediatric dosing: 0.25-5 mcg/kg/min IV, to a maximum dose of 20 mcg/kg/min

- Safety profile: Class C in pregnancy, Safety during lactation is unknown

- Nitroprusside: Reduces afterload.

- Is a more potent arteriolar vasodilator than nitroglycerin
- Poses the potential for thiocyanate toxicity
- If further afterload reduction is needed after administration of nitroglycerin, a nitroprusside drip can be started.

- Dosage: 0.3–10 mcg/kg/min
- Pediatric dosing: 0.25-4 mcg/kg/min to a maximum dose of 10 mcg/kg/min
- Safety profile: Class C in pregnancy. Possibly unsafe during lactation
- Nesiritide:
  - Has not been found to increase or decrease the rate of hospitalization or death. It might have a negative effect, causing hypotension. Its use in acute heart failure is still being investigated.
  - Dosing: 0.01 mcg/kg/min IV to a maximum of 0.03 mcg/kg/min
  - Safety profile: Class C in pregnancy. Safety during lactation is unknown
- Loop diuretics: Reduces preload.
  - Loop diuretics may be the first line of treatment for chronic heart failure. However, Cotter and colleagues found that for patients with acute heart failure accompanied by severe pulmonary edema and respiratory distress, high-dose nitro derivatives combined with a small dose of furosemide were more effective than high-dose furosemide with a small dose of nitro derivatives. IV dosing of diuretics, especially in large

doses, is more helpful than the oral route; diuresis will begin within 15 to 20 minutes after administration.

- The dose in emergency circumstances can be up to 2.5 times the patient's regular dose. If the patient is loop diuretic naïve; give 40 mg IV of furosemide or the equivalent of another loop diuretic.
- Check the safety profile for each individual medication that is considered or administered.
- Inotropics:
  - Severe left ventricular dysfunction or acute valvular problems may cause hypotension in some patients. Therefore, using the above agents can be harmful to these patients. Therefore, inotropic medications such as **dopamine and dobutamine** can be inevitable to preserve normal blood pressure.

## Procedure

### Bedside ultrasound

Please read **RUSH protocol** chapter and watch its' videos to learn more about US evaluation of pulmonary edema.

### Disposition Decisions

- All patients who present to an ED with acute heart failure and severe pulmonary edema should be admitted to either an

intensive care unit or an intermediate care unit based on the institution's ability to handle the necessary therapeutic modalities, especially drips and noninvasive positive-pressure ventilation (NIPPV).

- Some patients might be stable enough on a telemetry floor/ward, especially patients who had resolved symptoms and findings.
- Risk-stratifying patients with acute failure in the ED is difficult and requires additional investigation.

**References and Further Reading**, click [here](#)

# Aortic Dissection

---

by Shanaz Sajeed

## Case Presentation

*A 56-year-old male presented to the emergency department with sudden onset of severe tearing chest pain radiating to the back. He had a history of hypertension and hyperlipidemia. He was a smoker. Upon arrival, he appeared to be diaphoretic and in severe pain. He denied any prior history of chest pain. He had been without any infective symptoms lately. He was compliant with his medications, namely, amlodipine and simvastatin. At triage, his blood pressure was noted to be 80/60 mmHg with a pulse rate of 130 bpm. His oxygen saturation was 95% on room air, and his respiratory rate was 22 breaths per minute. On examination, he had muffled heart sounds, jugular venous distention, and radio-radial pulse delay.*

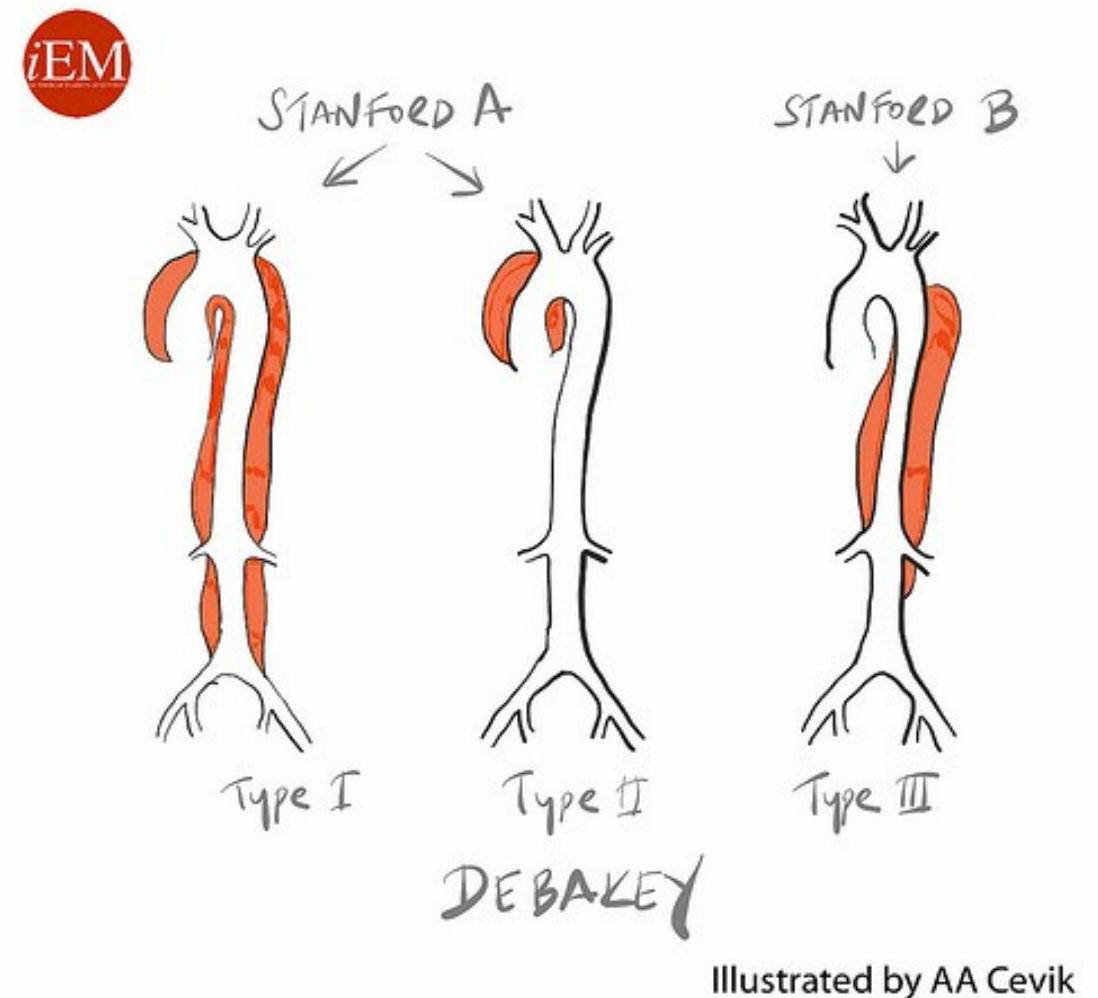


Audio is available [here](#)

## Introduction

Aortic dissection carries high morbidity and mortality. Although patients generally present with acute symptoms and classic signs, a subset of patients may present with syncope, GI bleeding, and neurological deficits. The clinician needs to remain vigilant for such atypical presentations. There are two standard anatomical classifications – Stanford and De Bakey. Stanford type A dissections (De Bakey I and II) involve the ascending aorta. Stanford type B (De Bakey III) dissections arise distal to the left subclavian artery (Illustration 4.1). Stanford A dissections are more common than Stanford B dissections (62% vs. 38%). The aortic wall consists of 3 layers – the intima, media and an outer layer known as the adventitia. Classic nontraumatic aortic dissection is usually due to a tear in the intimal layer of the aorta, leading to an intimal flap. The bridge between the media layer and the aortic lumen causes a subintimal hematoma. Then intima gets separated from the underlying media and adventitia. This false lumen of varying size may result in complete occlusion of major arteries that branch from the aorta leading to major ischemic complications such as limb ischemia, paralysis, stroke, renal failure as well as cardiac events.

**Illustration 4.1** De Bakey and Stanford classification of aortic dissection.



## Critical Bedside Actions and General Approach

The initial management of a patient with chest pain and hypotension warrants observation in a monitored area with continuous SpO<sub>2</sub> and cardiac documentation. The physician needs to assess airway, breathing, and circulation. A compromise in any of these necessitates immediate action. In this case



scenario, for example, the next priority would be to determine the etiology of this patient's hypotension and to initiate resuscitative and stabilizing measures. The physician should

- Administer supplemental oxygen and assess the patient's airway, breathing, and circulation to determine the need for any immediate critical interventions.
- Secure venous access by inserting two large bore IV cannulas into the antecubital fossa.
- In the hypotensive patient, administer an initial IV fluid bolus of 20 ml/kg crystalloid.

Note: A small initial bolus of fluid would not be harmful. Even in cardiogenic shock, it is likely to improve cardiac output and blood pressure transiently. Thus, one should not be hesitant to give an initial bolus of fluid in the hypotensive patient even if the etiology of shock is not immediately apparent.

## History Taking and Physical Examination Hints

### History Taking Hints

As initial resuscitation is going on, a focused history should be obtained at the bedside. Inquire about the site, onset, nature, duration, radiation as well as aggravating and relieving factors of the chest pain. Patients with acute Type A dissection classically present with :

- Sudden onset of 'ripping' or 'tearing' chest pain (85%) and/or interscapular back pain (46%)
- Pain is usually maximal at onset, unlike MI where pain usually gradually increases in intensity.
- Pain may migrate distally to the abdomen as dissection progresses
- However, a minority of patients may present atypically with abdominal pain (22%), syncope (13%) and stroke (6%).
- Patients often describe the pain as knife-like.

Other symptoms include dyspnea, dysphagia, focal weakness and altered mental status. Type B dissections present similarly. Occlusion of the main abdominal aortic branches may lead to mesenteric and solid organ ischemia. Patients may present with gastrointestinal bleeding, oliguria or anuria. Therefore, asking about gastrointestinal symptoms is an essential part of the history. Risk factors for aortic dissection should be assessed. These are;

- An aortic aneurysm
- Atherosclerosis
- Chronic Hypertension
- Coarctation of the aorta
- Congenital aortic valvular defects (e.g., bicuspid aortic valve)
- High-intensity weight lifting
- Increasing Age
- Infection leading to aortitis

- Inflammatory processes due to vasculitis. e.g., Takayasu's aortitis
- Inherited connective tissue disease (e.g., Marfan's, Ehlers Danlos Syndrome Type IV, familial forms of a thoracic aneurysm and dissection)
- Male gender
- Substance abuse such as cocaine, methamphetamine, MDMA

Past medical history findings that should prompt consideration of aortic dissection include:

- A family history of aortic dissection, aneurysm, or sudden death
- Chronic hypertension (most common predisposing factor)
- Documented aortic pathology
- History of tuberculosis or syphilis
- Known connective tissue disorder
- Previous cardiac surgery (especially valve repair) or vascular surgery

- Vasculitis

The physician should also assess the patient's anticoagulants use for increased bleeding risk, such as Warfarin/Novel Oral Anticoagulants or antiplatelet agents, which would increase their bleeding risk. It is also vital to elicit any specific allergies that may affect therapy or prohibit the use of IV contrast for imaging purposes. Elicit any relevant social history including questions about illicit drug use. Note: Cocaine use is associated with acute dissections.

### Physical Examination Hints

As initial stabilization and initial resuscitation take place, a focused clinical examination should simultaneously be performed. The physician should assess for signs of shock (e.g., cold extremities, delayed capillary refill, weak, thready pulse.) and aim to determine the etiology of hypotension if present. The physician should always consider bedside ultrasonography (RUSH protocol) to

facilitate diagnosis as soon as possible. Assess for pulse deficits. Pulse deficits are diminished or absent pulses caused by compression of the true lumen by the false lumen. Blood pressure difference between the left and right arm suggests aortic dissection. BP difference >20 mmHg between the two limbs is significant. Data analysis from the International Registry of Acute Aortic Dissection (IRAD) revealed that fewer than 20% of patients with proven acute aortic dissection had reported pulse deficits. The cardiopulmonary examination should focus on signs of cardiac ischemia, aortic insufficiency, cardiac tamponade and cardiogenic shock. When listening to the heart sounds, listen for:

- Distant/muffled heart sounds suggest pericardial effusion.
- Gallop rhythm, S3, S4 suggestive of heart failure.

- Diastolic murmur indicating aortic insufficiency. It is seen up to 75% of Type A dissections.

In the setting of the hypotensive patient, pulsus paradoxus and distended neck veins suggest cardiac tamponade. Examine for pulmonary findings of:

- Rales or Ronchi suggestive of heart failure
- Tachypnea and retractions indicating respiratory distress

Examine for signs of stroke or paraplegias/paresthesias suggestive of spinal cord infarcts due to occlusion of the spinal arteries. Examine the abdomen for pulsatile expansile masses suggestive of aneurysmal dilation. Signs of peritonitis may suggest mesenteric ischemia, a known complication of an aortic dissection.

## Differential Diagnosis

Aortic dissection should be considered in all patients with chest pain. The typical pain in acute thoracic aortic dissection is abrupt onset, severe and with radiation to the back. However, there is significant overlap between patients with myocardial infarction, and the two can be difficult to distinguish. A patient with aortic dissection may experience ischemic pain due to the involvement of the coronary arteries. There is a wide range of differential diagnosis in a patient with chest pain. Potentially life-threatening causes of chest pain include:

- Acute coronary syndrome
- Pericarditis/Myocarditis
- Pulmonary embolism
- Pneumonia/Pleural effusion
- Pneumothorax
- Rib fractures
- Esophageal rupture
- Mediastinitis

## Emergency Diagnostic Tests and Interpretations

### Bedside Tests

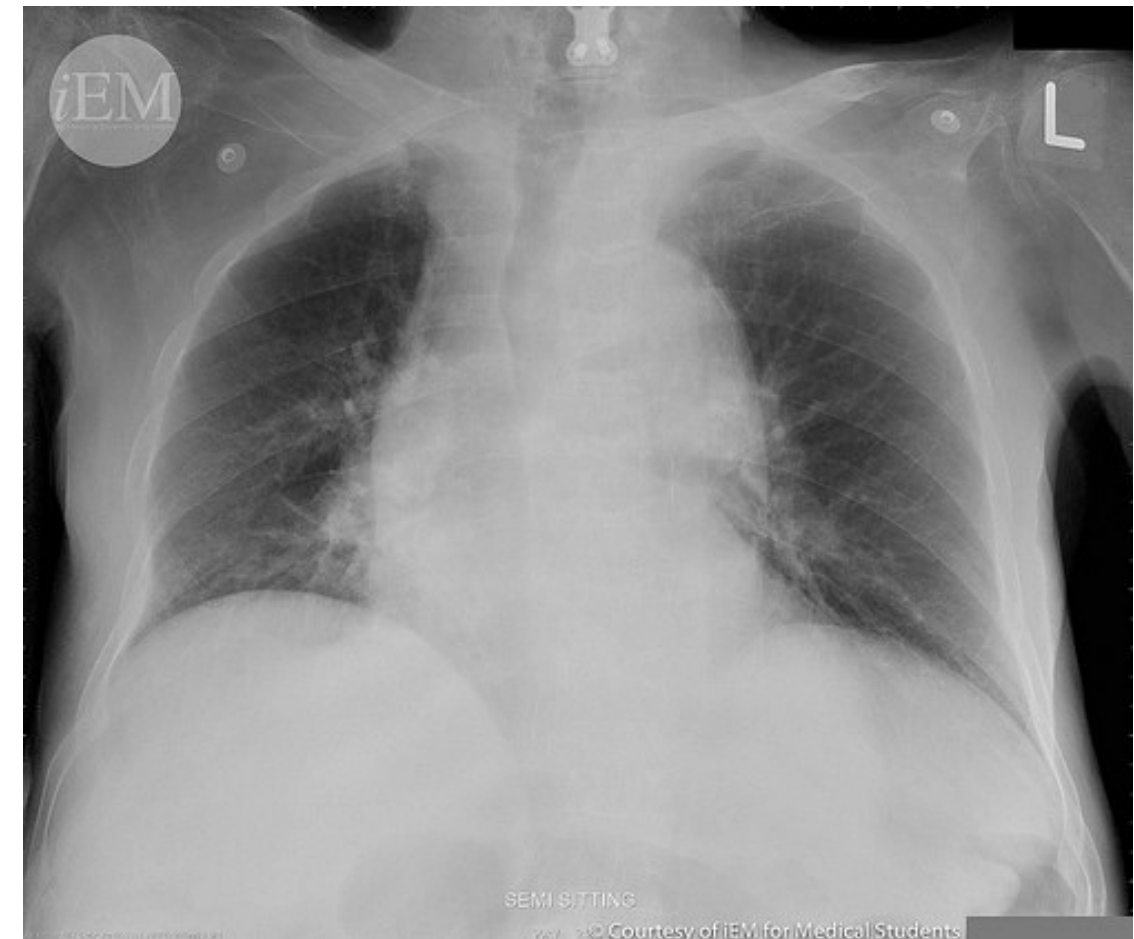
- Electrocardiogram(ECG) – may be normal, show nonspecific ST changes, or changes suggestive acute coronary syndrome. The most common coronary artery involved is the right coronary artery, leading to an inferior STEMI.
- Bedside transthoracic echocardiography may yield useful information such as the presence of pericardial fluid suggestive of tamponade. It can also give information on cardiac contractility. A dilated aortic root or dissection flap may be visualized on the parasternal long axis view.

- Chest X-ray – Abnormalities suggestive of dissection are present between 60-90% of cases.

#### These are;

- Depression of the left mainstem bronchus
- Displaced intimal calcification
- Indistinct or irregular aortic contour
- Left apical pleural cap
- Opacification of the “AP window” (i.e., clear space between the aorta and the pulmonary artery)
- Pleural effusion (left > right)
- Tracheal or esophageal deviation
- Widened aortic knob or mediastinum (present in only 63% and 56% of patients with type A and type B dissections, respectively).

**Image 4.6** Chest x-ray showing aortic dissection findings.



### Laboratory Tests

Blood investigations should include a full blood count, urea and electrolytes, coagulation, cardiac enzymes and crossmatch.

### Imaging Modalities

- Contrast-enhanced CT aortogram (Figure 2 and Figure 3) is usually the investigation of choice. In cases where CT poses a significant risk (e.g., pregnancy), MR Angiography of the aorta can be done.



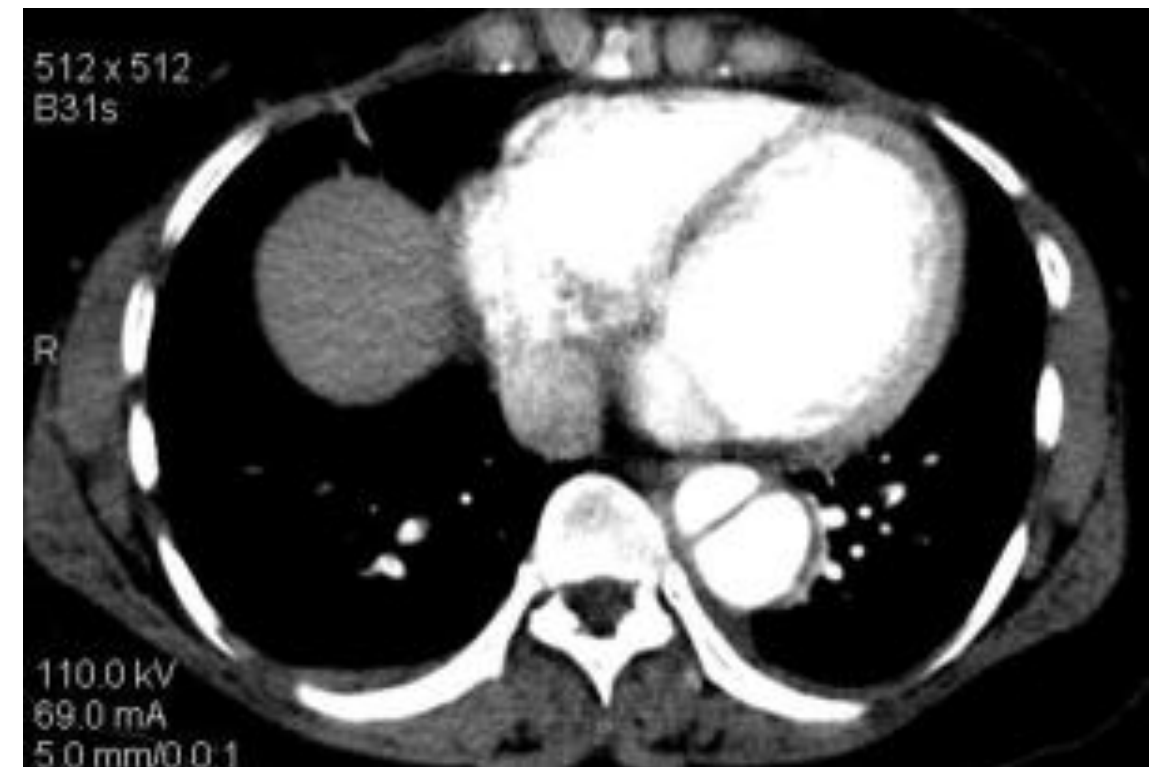
- Transesophageal echocardiogram (TEE) can be done at the bedside where there is a risk of contrast-induced nephropathy (Patients with impaired renal function) or contrast allergy, or in unstable patients.

**Image 4.7** CT scan - Stanford Type A dissection



*Type A dissection involving the ascending aorta demonstrating dissection flap as well as the true and false lumen. (Case courtesy of Dr. Frank Gaillard, Radiopaedia.org, rID: 8886)*

**Image 4.8** CT scan - Stanford Type B dissection



*Type B aortic dissection with dissection flap in descending aorta. (Case courtesy of Dr. Avni K P Skandhan, Radiopaedia.org, rID: 25409)*

## Emergency Treatment Options

Investigations and diagnostic workup should be done in parallel with the resuscitation of the patient. After initial assessment and stabilization and the definitive diagnosis made, reassess the patient and determine further management. Patients with aortic dissection may be hypotensive, normotensive or hypertensive.

### Hypotensive Patient

The priority is to maintain organ perfusion until definitive management:



- Administer IV crystalloid bolus of 20 ml/kg
- Consider vasopressors (if needed) to maintain a MAP: 70-80 mmHg
- If pericardial tamponade is present, emergent pericardiocentesis is indicated
- Blood transfusion is indicated if the hypotension is due to internal bleeding

## Hypertensive Patient

Aggressive blood pressure control is essential to reduce shear stress:

- Target a systolic pressure between 100-120 mmHg or MAP 70-80 mmHg. Heart rate should be between 60-80.
- IV  $\beta$ -blockers are the first-line therapy.
  - Labetalol: 20 mg IV slow injection, then 40-80 mg IV q10 min PRN, up to 300 mg IV total. An infusion of 0.5 to 2.0 mg/min can also be run.
- Calcium channel blockers such as diltiazem or verapamil may be used in patients with contraindication to  $\beta$ -blockers
- Sodium Nitroprusside may be added as adjunctive therapy for elevated blood pressure refractory to  $\beta$ -

blockers or Calcium Channel Blocker therapy. Avoid using as sole therapy as it can cause reflex tachycardia.

Practical Point: Hypotensive and drowsy patients need secured airway or intubation before any advanced imaging. Induction agents with cardiovascular stability are advised. Push dose vasopressors should be available in case of a precipitous drop in blood pressure. Investigations and diagnostic workup should be done in parallel with the resuscitation of the patient. Emergent surgical/interventional consult should be sought for definitive management.

- Type A dissections are usually managed surgically. The principal objectives are 1) relieve the symptoms, 2) reduce the complications, and 3) prevent aortic rupture and death. The affected layers of the aorta are sutured together, and the aorta is reinforced with a graft. Endovascular therapy is now becoming increasingly popular.
- Type B dissections are usually managed medically with aggressive blood pressure and pain control as well as continual monitoring for signs and symptoms of complications. Surgical management indications include:
  - Signs of bowel ischemia, limb ischemia or solid organ ischemia
  - Persistent pain

- Expanding hematoma or impending rupture
- Associated aneurysmal dilatation of the aorta

## Disposition Decision

Patients with acute aortic dissection should be managed in a high dependency or intensive care unit. The overall in-hospital mortality of aortic dissection is 27%. 30-day mortality of type A dissection with and without surgery is 26% and 58%, respectively. Type B dissection treated medically has 11% and surgically has 31% 30-day mortality.

**References and Further Reading**, click [here](#)

# Deep Vein Thrombosis (DVT)

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by Elif Dilek Cakal

## Case Presentation

*An 85-year-old woman, with a history of congestive heart failure, presented with right leg pain and swelling of 2 days' duration. She had been hospitalized for pneumonia one week earlier. Her vitals on arrival were: Blood Pressure: 138/84 mmHg, Pulse Rate: 65 beats per minute, Respiratory Rate: 14 breaths per minute, Body Temperature: 37°C (98.6°F), Oxygen Saturation: 96%. On examination, her right calf was reddish, tender, edematous and 4 cm greater in circumference than the left when measured 10 cm below the tibial tuberosity. Her Wells' Score for deep vein thrombosis (DVT) was 4 and suggested high-risk for DVT. Compression ultrasonography showed a thrombus in the popliteal vein. Enoxaparin (1 mg/kg, twice a day, SC) was started. No signs and symptoms of pulmonary embolism were observed. The patient was referred to a cardiovascular surgeon as an outpatient after discussion*



Audio is available [here](#)

*and confirmed understanding of discharge instructions.*

## Introduction

The annual incidence of DVT is 92 cases per 100000 persons. The rate steadily advances with increased age (32/100000 if age < 55 years, 282/100000 if age 65-74 years, 555/100000 if age >74). While 90% of DVT occurs in lower extremities, 10% of DVT occurs in upper extremities. Up to more than 40% of patients with lower extremity DVT have concomitant pulmonary embolism (PE), whether they may have related complaints or not.

## Critical Bedside Actions and General Approach

DVT is mostly a relatively benign disease; nevertheless, it may cause severe symptoms and limb- or life-threatening presentations. Emergency physician (EP) must check for signs of adverse outcome. Therefore patients should be evaluated for airway, breathing, circulation sequence and EPs try to understand possible immediate life-threatening problems. Concentrating on the patient focal complaint should be followed after the initial evaluation. Check vitals for instability and fever. Check for arterial pulses and signs of acute arterial thrombosis immediately in the case of every limp pain. Also, an extremely or entirely swollen limb indicates total or near total obstruction at a more proximal level. Increased compartment pressure may potentially disrupt the arterial flow. Diagnosing DVT in the emergency department (ED) is crucial. A timely started treatment may prevent the subsequent pulmonary embolism (PE) and chronic morbidities like chronic venous stasis and recurrent clots.

Some patients may ignore PE-related mild symptoms, or they may give priority to DVT-related ones. EP must concentrate on subtle PE-related sign and symptoms. In the spectrum of DVT, phlegmasia alba dolens, phlegmasia cerulea dolens and venous gangrene are vascular emergencies. They should be managed surgically, by endovascular interventions or thrombolytic treatment, in a time-sensitive manner. Upper extremity DVT has its own risk factors and consequences. It should be managed in its own context.

### Differential Diagnosis

Table 4.5 summarizes differential diagnoses of DVT. Unilateral and bilateral leg swelling and pain are two categories in order to differentiate the various causes. Bilateral leg swelling is more likely a clue for congestive heart failure, liver or renal failure, inferior vena cava compression than the bilateral DVT. However, patients symptoms and findings should be considered for ruling out these causes.

**Table 4.5** Differential Diagnoses of DVT

UNILATERAL LEG SWELLING AND/OR PAIN	BILATERAL LEG SWELLING AND/OR PAIN
Abscess/Necrotizing Fasciitis Arterial insufficiency Baker cyst rupture/inflammation Cellulitis Claudication Compartment Syndrome DVT Erythema Nodosum Hematoma Lymphangitis Lymphedema Musculoskeletal trauma Myositis Polyarteritis Nodosa Postphlebitic Syndrome Superficial thrombophlebitis Tendinitis/Achilles tendinitis Varicose Veins	Bilateral DVT Congestive heart Failure Inferior vena cava compression Liver Failure Renal Failure

*Please read Courtney DM. Venous Thrombosis. In: Adams JG, Barton ED, Collings JL, DeBlieux PMC, Gisondi MS, Nadel ES, editors. Emergency Medicine: Clinical Essentials. Philadelphia: Elsevier; 2013:611-617. and Ferri FF. Deep Vein Thrombosis. In: Ferri FF, et al, editors. Ferri's Clinical Advisor 2015: 5 Books In 1. Philadelphia: Elsevier; 2015:348-350. for more information.*



## History and Physical Examination Hints

- Neither medical history nor physical examination is specific to DVT. Clinical presentation may range from nearly asymptomatic to severely symptomatic or limb- or life-threatening.
- As a general rule, unilateral limb pain and swelling imply DVT.
- **Lower extremity DVT**
  - Unilateral leg pain and swelling are indicators of lower extremity DVT. Some patients may define fullness or cramping in the posterior aspect of the lower extremity. Bilateral symptoms are more likely in the course of other diseases. However, simultaneous bilateral DVT or obstruction of the inferior vena cava may cause bilateral symptoms.
  - Edema, redness, and tenderness are possible signs. None is specific. Homans sign refers to calf pain elicited by passive dorsiflexion of the

ankle. It is insensitive and nonspecific, therefore, useless.

- Because only history and examination are indeterminate, risk factors for DVT are essential to predict clinical probability. Known risk factors for DVT are as follows:
  - Previous history of PE or DVT
  - Recent Trauma or surgery
  - Cancer
  - Central or long-term vascular catheter
  - Age
  - Oral contraceptives
  - Hormone replacement therapy
  - Pregnancy
  - Immobility
  - Factor V Leiden mutation
  - Antiphospholipid antibody syndrome

- Prothrombin mutation
- Hyperhomocysteinemia
- Deficient levels of clotting factors
- Congestive heart failure
- Chronic obstructive pulmonary disease
- Air travel
- Obesity
- Phlegmasia alba dolens and phlegmasia cerulea dolens are vascular surgical emergencies. The features of these conditions are summarized in Table 4.6.

**Table 4.6** Surgical Emergencies Secondary To DVT

	PHLEGMASIA ALBA DOLENS	PHLEGMASIA CERULEA DOLENS
Appearance	Pale, cool, edematous (An example is available at <a href="https://www.thrombosisadviser.com/html/images/library/vte/deep-vein-thrombosis-right-leg-HR.jpg">https://www.thrombosisadviser.com/html/images/library/vte/deep-vein-thrombosis-right-leg-HR.jpg</a> )	Cyanosed, edematous, purple ecchymosis (An example is available at <a href="http://circ.ahajournals.org/content/125/8/1056/F1.expansion.html">http://circ.ahajournals.org/content/125/8/1056/F1.expansion.html</a> )
Distal pulses	Poor/Absent	Hard to palpate because of edema Absent if advanced
Pain	Positive	Positive
Mechanism	Massive iliofemoral venous thrombosis and associated arterial spasm	Arterial flow disruption due to venous congestion and increased tissue pressure
Thrombus location	In major veins (collaterals are generally spared)	In major veins and collaterals
Advances to	Phlegmasia cerulea dolens	Venous gangrene

*Treatment: IV Fluid + systemic anticoagulation + catheter-directed thrombolysis/systemic thrombolysis/surgical thrombectomy/mechanical thrombectomy.*

*Please read following references for more information (<http://lifeinthefastlane.com> accessed at 10.05.2016, <https://www.thrombosisadviser.com> accessed at 10.05.2016, and Mumoli N, Invernizzi C, Luschi R, Carmignani G, Camaiti A, Cei M. Phlegmasia Cerulea Dolens. *Circulation*. 2012; 125: 1056-1057.)*

#### • Upper extremity DVT:

- Upper extremity DVT is infrequent and accounts for approximately 10% of all DVTs. Its prevalence is increasing due to indwelling central catheters.
- Primary Upper extremity DVT is rare. A well-known form of primary upper extremity DVT is effort-related thrombosis named Paget-Schroetter syndrome. Paget-Schroetter syndrome generally occurs in otherwise healthy young men, after vigorous arm exercise or repetitive overhead activities. Patients with effort-related upper extremity DVT suggests an underlying venous thoracic outlet syndrome. In the absence of an obvious risk factor or underlying venous thoracic outlet syndrome, it is called idiopathic DVT.
- Catheter-associated DVT is the predominant secondary upper extremity DVT. Indwelling central venous lines, port systems and pacemaker or defibrillator are leading predisposing factors in descending order. Cancer, surgery, trauma, immobilization, pregnancy, oral contraceptive use and the ovarian hyperstimulation syndrome are the other predisposing factors for secondary upper extremity DVT.
- Severe upper extremity DVT may result in **superior vena cava syndrome**.

## Emergency Diagnostics Tests and Interpretation

- Approximately 90% of DVTs occur in lower extremities. Determination of pretest probability (PTP), D-dimer testing and bedside compression ultrasound are the milestones of management in ED.
- Wells' Criteria for DVT ([link](#)) stratifies patients according to their DVT risk. Scores  $\geq 2$  qualify a patient as "High Risk."
- A diagnostic algorithm is shown [here](#) (accessed at 10.05.2016)
- D-dimer is useful for its negative predictive value. When negative, it rules out DVT in the low-risk group. It does not confirm DVT when positive.
- Many ultrasound protocols for DVT are available. Related ultrasound videocasts can be found [here](#) (accessed at 10.05.2016)
- Upper-extremity DVT is diagnosed by Doppler ultrasonography.

**Watch** - A tutorial about diagnosing DVT with US.

**Watch** - Normal and Abnormal US findings for DVT

## Emergency Treatment Options

- The mainstay of medical therapy in ED is anticoagulation.
- Possible anticoagulation options are summarized in Table 4.7.

**Table 4.7** Medication For Anticoagulation in DVT

CLASS OF AGENT	DOSE	COMMENTS
Unfractionated heparin	80 U/kg IV bolus, then 18 U/kg/h IV infusion (Dose adjustment based on APTT)	Consider in inpatient therapy and in severe renal failure
<b>Low Molecular Weight Heparins</b>		
Dalteparin	100 IU/kg, twice a day, SC 200 IU/kg, once a day, SC	A standard treatment for DVT, preferred in outpatients as a first line therapy if not contraindicated
Enoxaparin	1 mg/kg, twice a day, SC 1.5 mg/kg, once a day, SC	A standard treatment for DVT, preferred in outpatients as a first line therapy if not contraindicated
Tinzaparin	175 IU/kg, once a day, SC	A standard treatment for DVT, preferred in outpatients as a first line therapy if not contraindicated
<b>Factor Xa inhibitors</b>		
Fondaparinux	< 50 kg - 5 mg, once a day, SC 50-100 kg - 7.5 mg, once a day, SC > 100 kg - 10 mg, once a day, SC	Do not use in renal failure

Please read <http://emedicine.medscape.com> (accessed at 10.05.2016) for more information.

The indications for more advanced therapies like catheter-directed thrombolysis, percutaneous mechanical thrombectomy, conventional surgery or systemic thrombolysis are as follows:

- Phlegmasia cerulea dolens
- Inferior vena cava thrombosis
- Subacute and chronic iliofemoral DVT
- Acute iliofemoral or femoropopliteal DVT

Though all are useful, endovascular interventions are preferred over more invasive interventions in capable centers so as to minimize the consequent risks. (Bleeding or perioperative complications, etc.)

The pain medication is advised for patients who are suffering from severe pain.

## Pediatric, Geriatric, Pregnant Patient and Other Considerations

### Pediatric Considerations

DVT is infrequent in children and almost always associated with risk factors. Central venous catheter-associated upper extremity DVT is relatively common in children. LMWH is the mainstay of the therapy.

- Enoxaparin:

- <2 months: 1.5 mg/kg/ dose SC, twice a day
- >2 months: 1.0 mg/kg/dose SC, twice a day

### Geriatric Considerations

DVT management does not alter in the elderly. Frequency and severity of DVT increase. Anticoagulation complications are more frequent than younger counterparts. Concomitant diseases and possible drug interactions complicate the management.

### Pregnant Patients

DVT management does not alter in pregnant. Pregnant women are susceptible to DVT. LMWHs are the drug of choice during pregnancy. All pregnant patients with DVT should be admitted to hospital.

### Patients With Isolated Calf Vein Thrombosis

The need for treatment is controversial.

## Disposition Decisions

### Admission Criteria

Most patients with DVT can be treated as outpatients. EP can decide the patients that need admission based on four questions ([link](#)).

1. Does the patient have massive DVT?
2. Does the patient have symptomatic **pulmonary embolism**?

3. Is the patient at high risk for anticoagulant-related bleeding?
4. Does the patient have major comorbidity or other factors that warrant in-hospital care

One or more positive answers should lead EP to admission.

**Consider admission if any is present:**

- Suspected or proven concomitant PE
- Significant cardiovascular or pulmonary comorbidity
- Iliofemoral DVT
- Contraindications to anticoagulation
- Familial or inherited disorder of coagulation
- Familial bleeding disorder
- Pregnancy
- Morbid obesity (>150 kg)
- Renal failure (creatinine >2 mg/dL)
- Unavailable or unable to arrange close follow-up care
- Unable to follow instructions
- Homeless patient

- No contact telephone
- Geographic location (too far from the hospital)
- Patient/family resistant to outpatient therapy

**Discharge Criteria**

All patients lacking admission criteria may be treated as outpatients after a confirmed understanding of discharge instructions. Several discharge instructions are available [online](#).

Referral: Patients must be referred to cardiovascular surgeons.

**References and Further Reading**, click [here](#)



# Hypertensive Emergencies

---

by Sadiye Yolcu

## Case Presentation

*A 68-year-old man with tearing chest pain presented to the emergency department. He had a history of coronary artery disease and hypertension. BP: 220/160 mmHg, HR: 105 bpm, RR: 20/min, T: 37, SpO2: 96% in room air. In the initial evaluation, airway and breathing were intact. Diastolic murmur was heard on cardiac auscultation, and pulses were positive in all extremities. He has a normal mental state (GCS 15) and no lateralized motor deficit. A difference in systolic blood pressure was measured between upper extremities (220/160 vs. 180/140 mmHg). ECG showed nonspecific ST-T changes and sinus tachycardia.*



Audio is available [here](#)

## Introduction

Systemic hypertension is a common medical problem. It affects over 1 million people worldwide. ER clinicians commonly encounter this problem. Rapid diagnosis, evaluation, differentiation of hypertensive emergencies and hypertensive urgencies, and appropriate treatment of these conditions are required to prevent morbidity and mortality.

The levels above 180 systolic BP and 110 diastolic BP are considered very dangerous which may cause end-organ damage such as intracranial bleeding, aortic dissection, renal failure, etc. Having end-organ damage is the hypertensive emergency. Having high blood pressure without any signs of end-organ damage is the hypertensive urgency. Retinal hemorrhage or exudates/papilledema associated with hypertension is defined as malignant hypertension.

Hypertensive emergencies require action within one hour to abolish the risks of developing complications. Hypertensive

urgencies are defined as situations requiring actions within 24 hours and yet do not compromise the risk of developing complications within that period.

### Hypertensive emergencies include

- Acute aortic dissection
- Acute coronary syndrome
- Acute heart failure
- Acute renal failure
- Eclampsia
- Hypertensive encephalopathy
- Intracerebral/subarachnoid hemorrhage
- Pheochromocytoma,
- Sympathomimetic drug use (cocaine etc.),
- Stroke

### Hypertensive urgencies include

- Diastolic tension  $\geq 140$  mmHg without complication

- Malign hypertension without complication

- Perioperative hypertension
- Pheochromocytoma,
- Sympathomimetic drug use (cocaine, etc.)

## Critical Bedside Actions and General Approach

The priority should be given to initial stabilization of the patient (C-A-B) as other critically ill patients. Depending on patients' symptoms in addition to high blood pressure, the cardiac monitorization, oxygen (if necessary), two large bore IV access should be established and blood samples (CBC, BUN, Cr, coagulation, cardiac markers, type, and cross-match) sent to the laboratory. ECG and chest x-ray should be ordered.

Lowering BP should be balanced with the level of BP, patient's symptoms as well as harm-benefit situation.

## Differential Diagnosis

The most critical step in the differential diagnosis is the definition of the hypertensive situation (emergency or urgency). Suspicion of hypertensive emergencies aligns with hypertension and end-organ damage. Depending on patient symptoms and findings, hypertensive emergencies differentials include severe problems such as intracranial hemorrhage, ischemic stroke, aortic dissection, acute MI, AAA rupture, heart failure, renal failure, limb or organ ischemia, etc. In addition to these end-organ damages, other differentials (seizure, brain tumor, encephalitis, encephalopathy, drug overdose, etc.) should also be considered.

## History and Physical Examination Hints

The previous medical history of the patient (chronic diseases, antihypertensive drugs usage, previous end-organ compromise, etc.) should be taken. Chest pain for myocardial infarction, aortic dissection, dyspnea for pulmonary edema, headache, mental status, seizure for hypertensive encephalopathy should be asked.

The patients present mostly with ischemic stroke, pulmonary edema, hypertensive encephalopathy, or congestive heart failure. Therefore, history and physical exam should be focused on these problems during the initial and secondary evaluation. In the physical examination, measure the blood pressure from both arms and assesses the patient for end-organ compromise (neurologic-ophthalmologic-cardiac).

Each of these hints was given in the specific disease chapters. Therefore, we advise you to review those chapters too.

## Emergency Diagnostic Tests and Interpretation

An electrocardiogram (ECG) and chest X-ray should be performed. ECG may show arrhythmias, nonspecific ST-T changes or obvious acute MI findings. The chest x-ray may give hints about aortic dissection, aneurysm, pulmonary edema.

What is your opinion about the chest x-ray (Image 4.9)?

Bedside ultrasonography may help to diagnose some critical pathologies timely. These are pulmonary edema, aortic aneurysm or dissection, heart failure, and increased intracranial pressure.

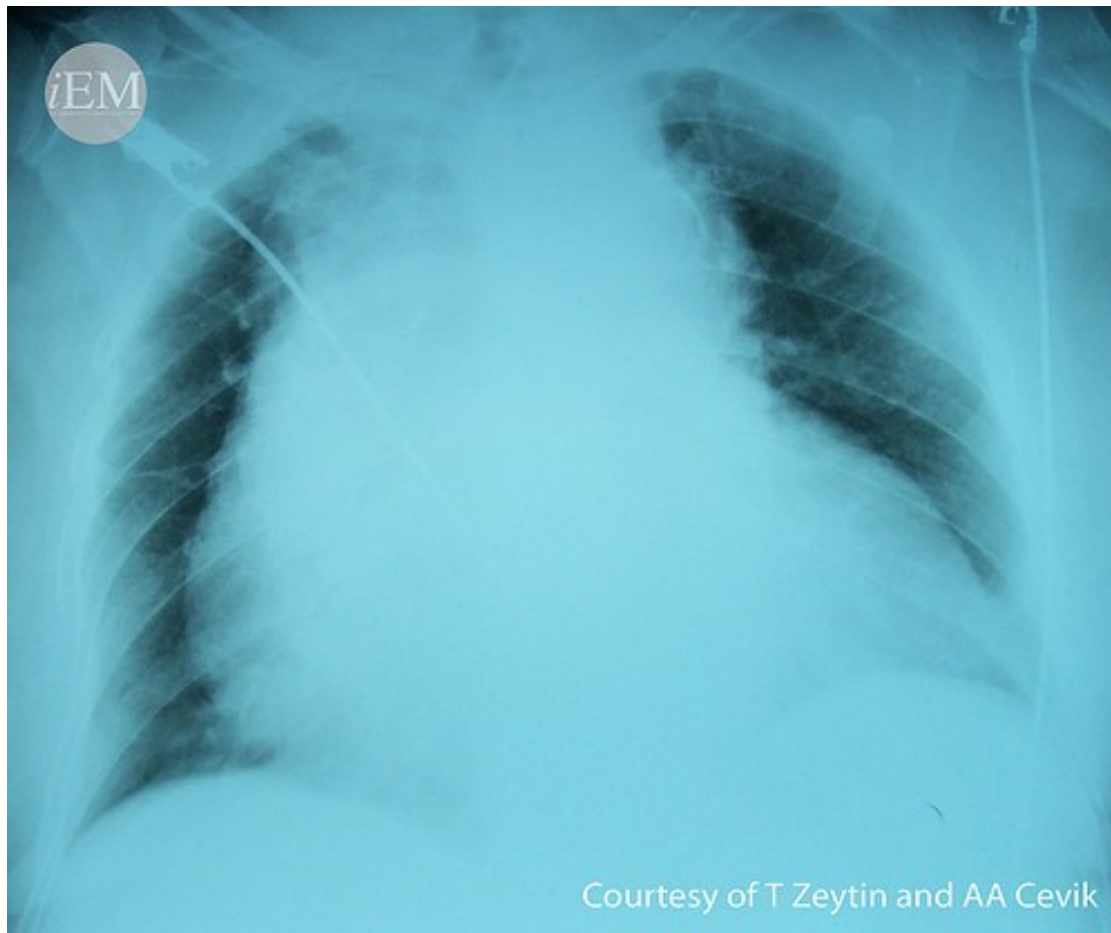
What is your opinion about the transthoracic ultrasound [here](#)?

Blood urea nitrogen (BUN), electrolytes, complete blood count (CBC), liver-renal function tests, coagulation parameters, cardiac enzymes and urine analyses should be checked. BUN and Cr may show renal impairment. Hematuria and proteinuria in the urine should also be checked.

Some patients may require further investigations with CT or MRI depending on their symptoms and findings.

What is your opinion about the CT (Image 4.10)?

Image 4.9

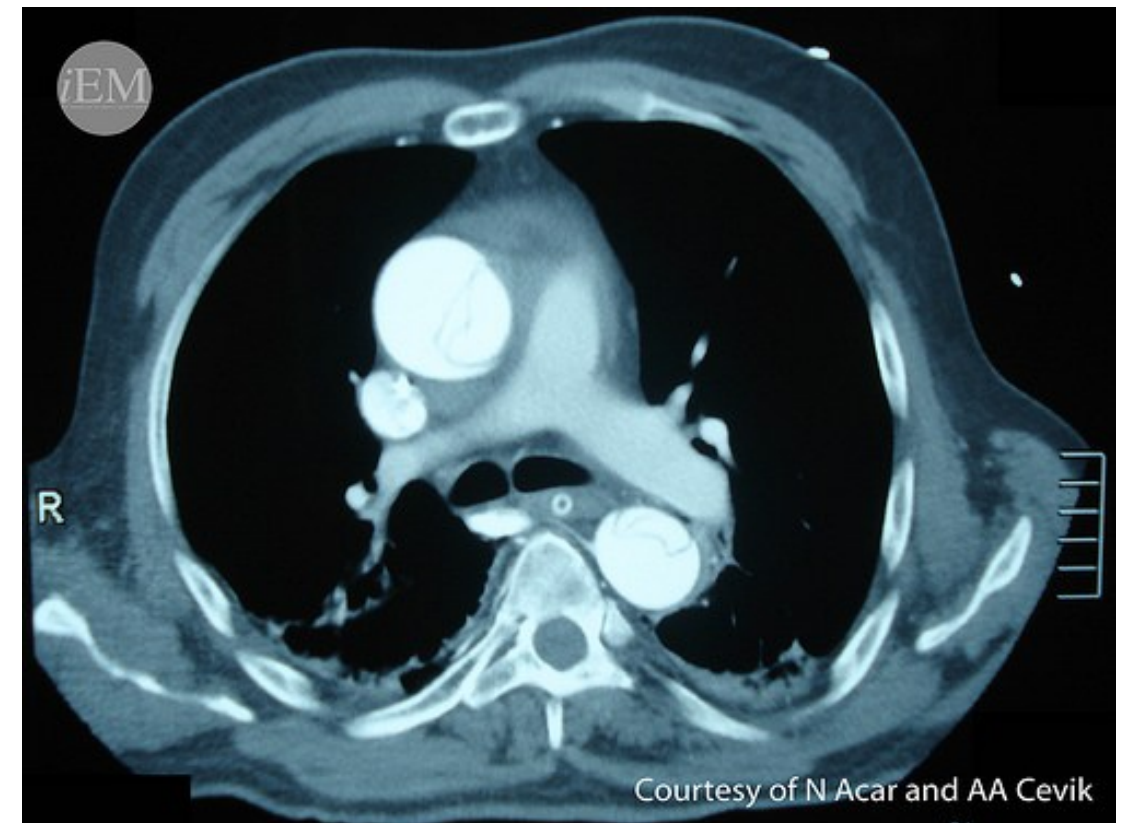


## Emergency Treatment Options

### Initial Stabilization

Support C-A-B and stabilize the patient as needed. Cardiac monitoring, pulse oximetry, oxygen administration, and IV access required for all hypertensive emergency cases. Key precaution in the control of hypertensive situations is to maintain the balance of the benefits of immediate decreases in BP against the risk of a significant decrease in target organ perfusion. Therefore, IV agents are preferred because of their titration option. Do not

Image 4.10



ignore pain medication because some of them require effective pain control.

### Medications in specific problems

#### Aortic dissection

The aim is to reducing shearing forces by decreasing the heart rate to 60-80 beats/min, and the systolic pressure to 140 mmHg and below, then to 120 whether the patient can tolerate. Organ perfusion should be monitored carefully. Na nitroprusside (0.3-0.5  $\mu\text{g/kg}$ ) is a potent agent, and the dose can be arisen by 0.5  $\mu\text{g/kg/min}$  each time till the maintaining the expected effect on blood



pressure. Along with Na nitroprusside, Esmolol (300 µg/kg IV bolus, then 50 µg/kg /min infusion) or labetalol (20-40 mg IV, then 20 mg IV on every ten mins, the maximum dose is 300 mg) helps to control heart rate. If beta blockers are contraindicated, verapamil (5-10 mg IV or diltiazem 0.25 mg/kg IV can be used.

### **Acute Hypertensive Pulmonary Edema**

The blood pressure shouldn't be decreased by more than 20-30%. The first choice is nitroglycerin (5-100 µg/min IV infusion). Start with 5 µg/min; then it can be increased up to 200 µg/min by increasing 10 µg on every five mins. Enalaprilat (0.625-1.25 mg IV in 5 mins every 4-6 hours) and nicardipine 5 mg/hr IV infusion, if no control in 15 mins 2.5 mg/hr dose can be added on every 15 mins).

### **Acute Coronary Syndrome**

Maximum 20% of the blood pressure should be acutely decreased if the systolic blood pressure is higher than 160

mmHg. Nitroglycerin or oral metoprolol (50-100 mg/12 hrs or IV 5mg on every 5-15 mins up to 15 mg)

### **Acute Sympathetic Crises**

Benzodiazepines are the initial treatment. Nitroglycerine can be considered if benzodiazepines are not effective. Phentolamine is another choice (5-15 mg IV).

### **Acute Renal Failure**

The blood pressure decreased up to 20% if it is higher than 180/110 mmHg. Nicardipine, labetalol, or fenoldopam is recommended agents.

### **Intracerebral Hemorrhage**

The mean arterial pressure (MAP) should be decreased to 130 mmHg if the patient has increased intracranial pressure findings. If no suspicion of increased intracranial pressure, the MAP can be decreased to 110 mmHg or the systolic blood pressure to 150-160 mmHg. Esmolol and labetalol can be used.

### **Subarachnoidal Hemorrhage**

The systolic blood pressure and the MAP should be lower than 160 mmHg and 130 mmHg, respectively. Esmolol and nicardipine can be used.

### **Ischemic Stroke**

If the fibrinolytic will be used, the systolic blood pressure should be lower than 185/110 mmHg. If the patient will not take a fibrinolytic treatment, then it is important to maintain the BP lower than 220/120 mmHg. Nitroglycerin and nicardipine can be used.

### **Hypertensive Encephalopathy**

The first agent is Na nitroprusside and followed by labetalol, nicardipine, fenoldopam. The systolic blood pressure shouldn't be decreased by more than 25% of the total. A 160-170 mmHg systolic blood pressure is expected in first 2-3 hours.



## Asymptomatic Situations

Oral antihypertensives (hydrochlorothiazides 25 mg/day, Metoprolol 25 mg/day, angiotensin receptor blockers, ACE inhibitors) should be given in the ED and prescribed to the patients whose systolic blood pressure is higher than 180-200 mmHg and the diastolic blood pressure higher than 110/120 mmHg.

## Pediatric, Geriatric, and Pregnant Patients

In pregnant patients who have underlying hypertension may present with severe preeclampsia, stroke, pulmonary edema, fetal decompensation, etc. IV hydralazine and oral nifedipine are equally effective in pregnant patients. In the pediatric population, the hypertensive emergency with end-organ effects requires immediate, and gradual decreasing of the BP. Metoprolol is effective and safe in the pediatric population.

## Disposition Decisions

### Admission Criteria

All patients with hypertensive emergencies, signs of end-organ damage are admitted to the intensive care or high dependency care unit.

## Discharge Criteria

Hypertensive urgencies (Absence of end-organ damage symptoms and findings, known to have hypertension, reversible causes, etc.)

## Referral

Patients should refer to their primary care physician or hypertension clinic in 7 days.

**References and Further Reading**, click [here](#)

# Pulmonary Embolism

---

by Elif Dilek Cakal

## Case Presentation

*A 45-year-old female with no prior medical history presented to the emergency department (ED) with three days of constant shortness of breath. She was suffering from left-sided sharp chest pain, which is stronger during inhalation. She had felt breathless while she was climbing upstairs during the previous week. She had no cough or expectoration. She was a non-smoker; her only drug was daily oral contraceptive. Vitals at arrival were as follows: Blood Pressure: 116/72 mmHg, Pulse Rate: 102 beats per minute (bpm), Respiratory Rate: 18 breaths per minute, Body Temperature: 37°C (98.6°F), Oxygen Saturation: 95%. Physical examination revealed no abnormality except for the left-sided basilar crackles. Chest X-ray was unremarkable. The emergency physician (EP) proceeded to investigate differential diagnoses. Her Well's Score for pulmonary embolism was 4.5 (moderate) because of*



Audio is available [here](#)

*increased heart rate and lack of alternative diagnosis. The laboratory results showed negative  $\beta$ -HCG, normal renal function test, platelet number and a D-dimer measurement of 751 ng/ml (cutoff = 550 ng/ml). EP explained these results to the patients and suggested a computed tomographic pulmonary angiography (CTPA). CTPA showed filling defects within the left pulmonary artery, left anterior and lateral segmental artery associated with pulmonary embolism. Enoxaparin, 1.0 mg/kg, twice a day (80 mg = 0.8 ml, each dose for approximately 80 kg patient), was started. Her Pulmonary Embolism Severity Index (PESI) was 65 (class I) and implied a very low risk. As a shared decision with the patient and respiratory physician, the patient was referred to the respiratory physician as an outpatient after discussion and confirmed understanding of discharge instructions.*

## Introduction

The incidence of pulmonary embolism (PE) is approximately 1.5 new cases per 1000 persons. Patients with chest pain, shortness of breath and syncope should have pulmonary embolism excluded. Atypical presentations include mental deterioration in patients with prior dementia. EP must maintain a high index of suspicion as the potential outcome of a misdiagnosis is catastrophic. The mortality of untreated PE is estimated to be 30% whereas the all-cause 30-day mortality of diagnosed PE is only 8%.

## Critical Bedside and General Approach

First, the EP must determine whether the patient is stable or unstable. Instability and shock warrant stabilization in addition to simultaneous diagnostic and therapeutic effort. If the patient is hypoxic, administer oxygen. Severe hypoxemia or mental deterioration necessitate intubation. If the patient is hypotensive, administer only 500 mL IV bolus saline. Aggressive IV fluid may increase the right heart strain and shock. If hypotension persists, give IV vasopressors, particularly norepinephrine or epinephrine.

Obtain an ECG to exclude STEMI and dysrhythmia. Perform a thorough bedside ultrasound. EP can rapidly exclude pericardial tamponade, pneumothorax, and intraabdominal bleeding via bedside ultrasound. Right ventricular enlargement or the presence of deep venous thrombosis (DVT) gives hints of pulmonary embolism (please check RUSH Protocol chapter).

What is your diagnosis in US given [here](#)?

- If the patient is stabilized, the patient should directly undergo CTPA.
- If the patient remains unstable or CTPA is unavailable, bedside ultrasound is the only diagnostic tool.
- If CTPA confirms or bedside ultrasound strongly suggests pulmonary embolism, thrombolysis is indicated.
- If the patient is stable with high suspicion of PE, but the diagnostic measures are expected to delay, administering of one dose low molecular weight heparin (LMWH) is recommended.

## Differential Diagnosis

Potentially life-threatening differential diagnoses of pulmonary embolism are summarized in Table 4.8.

Non-Life-Threatening Causes are Bronchitis, Chest wall pain/Costochondritis, Pleuritis/Pleurisy, GI Abnormalities (GERD, Peptic Ulcer, Gastritis), Panic Attack/Anxiety Disorder, Rib Fracture.

## History and Physical Examination Hints

- The character and severity of the clinical presentation may vary tremendously from being asymptomatic to sudden death. The patient's prior condition, clot's size, and localization affect

presentation. Even highly qualified EPs may miss the diagnosis because of vague signs. Previously healthy young patients tend to be mildly symptomatic with normal vital signs. Prior cardiopulmonary disease and cognitive dysfunction generally obscure the diagnosis. Therefore, atypical presentations are frequent in elderly patients. Proximal clots cause dyspnea via ventilation-perfusion mismatch. Pneumonia-like presentation and pain due to pulmonary infarction are more often in distal clots. The presence or absence of sudden onset symptoms neither increase nor decrease the probability of PE. Fewer than half of patients describe sudden onset.

- Most patients with PE complain of dyspnea (82-85%), chest pain (40-49%), pre-syncope or syncope (10-14%), and hemoptysis (2%). Other PE-related signs and symptoms include functional or mental deterioration, arterial hypotension, cough, flank pain, abdominal pain, dizziness, light-headedness, tachypnea (30-60%), fever, diaphoresis, and anxiety. DVT-related symptoms may accompany. Some cases are asymptomatic and diagnosed incidentally.
- Shortness of breath, vague or apparent, is the most common symptom. A patient with PE typically presents with 2 to 3 days of new-onset shortness of breath that is not explained by a known medical condition, now worsened enough to seek care. Because the embolic burden is loading gradually, most patients describe dyspnea on exertion days to weeks before dyspnea at

**Table 4.8** Potentially Life-Threatening Differential Diagnoses Of Pulmonary Embolism

POTENTIALLY LIFE-THREATENING CAUSES	HISTORY AND PHYSICAL EXAMINATION	BED-SIDE ACTIONS	IMAGING	LABORATORY	COMMENTS/ PEARLS AND PITFALLS
ACS – STEMI	Typical chest pain	<b>ECG</b>		Troponins	Central PE causes angina-like chest pain and cannot be excluded only based on the nature of pain.
ACS – NSTEMI	Typical chest pain	ECG		Troponins	Troponins may be elevated in PE.
Cardiogenic Shock/ Congestive Heart Failure	History of CHF	<b>Bed-side ultrasound (e.g. RUSH protocol)</b>	Pulmonary edema on chest X-ray	BNP Pro-BNP	BNP and pro-BNP may be elevated in PE.
Cardiac Dysrhythmias	Personal/Family history of cardiac dysrhythmias, new-onset “convulsions”	<b>ECG</b>			
Pneumothorax	Trauma?	<b>Bed-side ultrasound</b>	Pneumothorax on chest X-ray		
Cardiac Tamponade	Malignancy?	<b>Bed-side ultrasound</b>			Malignancy increases both PE and cardiac tamponade incidence.
Pneumonia	Cough with sputum, fever, immunosuppression	Bed-side ultrasound	Pneumonic infiltration on chest X-ray	WBC CRP Procalcitonin	
Esophageal Rupture			Chest X-ray CT/CTPA		
Pulmonary Malignancy	History		Chest X-ray CT/CTPA		Malignancy increases cardiac tamponade incidence.
Asthma	History Bronchospasm on examination				
Aortic Dissection		Bed-side ultrasound	<b>CT/CTPA</b>		
Pericarditis/Myocarditis	History of flu-like symptoms	<b>ECG</b> <b>Bed-side ultrasound</b>		Troponins	

The most helpful diagnostic ways to establish the diagnoses are in bold. Original by author.



rest.

- Contrary to common misbelief, PE may cause both pleuritic and angina-like chest pain. Distal emboli induce atypical, pleuritic, stabbing-like chest pain due to pleural irritation. Central emboli may present as typical angina-like chest pain, possibly associated with RV ischemia. Thus, exclusion solely based on the quality of chest pain is impossible.
- The frequency of syncope and pre-syncope among the ED patients with confirmed PE remains highly variable in different studies (4-22%). On the other hand, only in a minor group of patients presented with pre-syncope and syncope, the final diagnosis is PE. Yet, patients with PE who present with syncope tend to have major PE.
- Haemoptysis is not common but is more specific to PE.
- Vital signs are variable. Most patients have relatively normal vitals. Some are in shock and shock is a predictor of bad outcome. Heart rate > 100 bpm and oxygen saturation <95% increase the probability. Fever does not exclude PE, though an oral temperature >39.2°C (102.5°F) greatly decreases the possibility. Mild or severe increase in respiratory rate may be present. Normalization of vital signs with treatment or time does not change the likelihood of PE.
- No single examination sign confirms or excludes PE. DVT-related signs increase the possibility. Pulmonary infarction

secondary to PE or other diagnoses may cause crackles. Bronchospasm primarily dictates other diagnoses. However, the EP must consider that underlying PE exacerbates Chronic Obstructive Pulmonary Disease (COPD). Treatment-resistant COPD exacerbations may imply PE.

- The combination of history and physical examination is frequently insufficient to diagnose. Thus, the EP must investigate risk factors to determine the likelihood of PE. The risk factors in the emergency setting differ from the general population or longitudinal risk factors.

**Table 4.9** Selected Risk Factors of Pulmonary Embolism In The ED Setting

INDICATORS OF PE IN THE ED SETTING	MAY BE LESS SIGNIFICANT IN THE ED POPULATION
Age > 50 Recent Surgery Recent Major Trauma Immobilization Estrogen Prior VTE Postpartum Inherited Thrombophilia Active cancer	Pregnancy Smoking Family History of VTE Inactive cancer Travel

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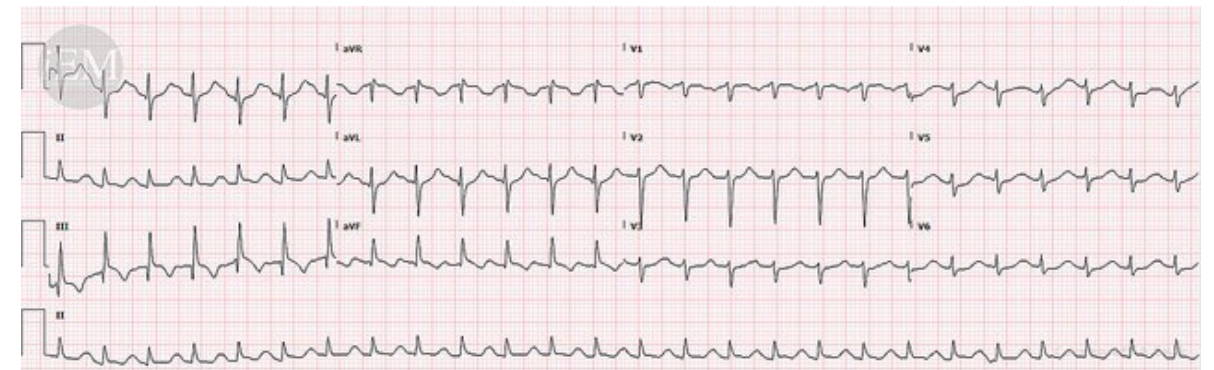
Table 4.9 summarizes selected common risk factors in the ED setting. For a more detailed discussion, please refer to the relevant sections of “Emergency Evaluation For Pulmonary Embolism, Part 1: Clinical Factors That Increase Risk” at this [link](#) and “Clinical Features From the History and Physical Examination That Predict the Presence or Absence of Pulmonary Embolism in Symptomatic Emergency Department Patients: Results of a Prospective, Multicenter Study” at this [link](#).

- Up to 30% of adult patients are without risk factors at the time of the diagnosis.
- Clinical prediction rules stratify patients according to their pretest probabilities.
  - Wells’ Criteria for PE – [link](#)
  - Revised Geneva Score (RGS) – [link](#)
- Experienced physician’s gestalt and clinical prediction rules showed similar performance in some studies, but the use of clinical prediction rules are strongly recommended for inexperienced physicians.
- Pulmonary Embolism Rule-Out Criteria (PERC) is recommended for the bedside exclusion of low-risk patients – [link](#)

## Emergency Diagnostic Tests and Interpretation

- Bedside ECG showstachycardia and non-specific ST-T changes in most of the cases. Acute S1Q3T3 finding in the ECG is seen only increased right ventricle enlargement and pressure which seen massive emboli. These finding can also be seen in core pulmonale.

**Image 4.11** ECG shows S1Q3T3



- Pulmonary embolism should come to mind in a large number of patients because of the changeable and vague nature of its presentation. Proper assessment of clinical probability, D-dimer testing and CTPA are cornerstones of management. Other diagnostic tools like lung scintigraphy, bedside echocardiography and compression venous ultrasonography of bilateral lower extremities may prove useful in special circumstances such as pregnancy, unavailability of CTPA, instability or shock.

What is your diagnosis in CT given [here](#)?

The high frequency of patients with symptoms implying PE results in the dilemma of underdiagnosing or overtesting for PE. Underdiagnosing PE increases mortality and morbidity. Overtesting raises emergency medicine crowding and potential complications due to chosen diagnostic modality. A true understanding and strict application of proven and reliable clinical rules and algorithms are recommended.

- The first step is the evaluation of the patient's stability. The diagnostic and therapeutic measures in stable and unstable patients are different. The presence of shock or hypotension immediately indicates high-risk PE while the absence of those implies non-high-risk PE. The proposed algorithms for both are as follows:

- Non-high-risk PE: The clinical approach for stable patients is demonstrated at this [link](#) as a part of "Emergency Evaluation For

Pulmonary Embolism, Part 2: Diagnostic Approach.

- High-risk PE: The clinical approach for unstable patients is demonstrated at this [link](#) as part of 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism.
- Once PE is confirmed accordingly, prognostic assessment is the next step. Clinical parameters and right ventricular function via imaging and biomarkers help the EP to predict prognosis.
  - The mostly studied prognostic prediction rules are Pulmonary Embolism Severity Index (PESI) – [link](#). and its simplified version (sPESI) – [link](#).
  - Right ventricular dysfunction has been reported  $\geq 25\%$  of patients. It implies a low cardiac output and adverse outcome. Echocardiographic findings of right ventricular dysfunction include right ventricular

dilatation and hypokinesis, septal flattening and paradoxical septal motion, diastolic left ventricular impairment. Other findings include direct visualization of pulmonary embolism, pulmonary arterial hypertension, right ventricular hypertrophy and patent foramen ovale. A normal right ventricular function does not exclude PE.

- Elevated brain natriuretic peptide (BNP), N-terminal (NT)-proBNP, troponins, creatinine, and D-dimer predict higher mortality. Negative biomarkers and D-dimer indicates a good prognosis.
- Patients are classified into four groups based on early mortality risk. Patients who are hypotensive or in shock are defined as high-risk regardless of other evaluations. Normotensive patients who are PESI class III-IV or sPESI class  $\geq I$  are at intermediate risk. Intermediate risk group divides into intermediate-high

risk and intermediate-low risk groups, according to RV function and cardiac biomarker values. Lastly, a PESI class I-II or sPESI class 0 normotensive patient is defined as a low-risk patient. The proposed risk stratification ([link](#)) and risk-adjusted management strategies in acute PE is demonstrated at this [link](#) as part of 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism.

## Emergency Treatment Options

### Initial Stabilization

Stabilizing interventions, diagnostic and therapeutic effort must begin immediately and continue till admission for an unstable patient. Development of shock, hypotension or hypoxemia in the course of ED stay warrants prompt stabilization.

- If the patient is hypoxemic
  - Administer oxygen

- Intubate, if necessary. Beware of high intrathoracic pressure for it may worsen the right ventricular failure. Therefore; aim:
  - Low tidal volumes (about 6 mL/kg lean body weight)
  - limited positive end-expiratory pressure
  - to keep end-inspiratory plateau pressure < 30 cm H<sub>2</sub>O
- Use non-invasive mechanical ventilation with caution.
- Mental deterioration and coma may dictate intubation.
- If the patient is hypotensive
  - Give 500 mL normal saline IV bolus. Avoid excessive IV fluids for it may increase right ventricular strain.
  - If fluid bolus does not help, start vasopressors. Norepinephrine and epinephrine are preferred over

dobutamine/dopamine, except for a selected group of patients with known congestive heart failure.

- Confirm PE with whether CTPA or bedside ultrasound as the patient's status permits. Prepare for thrombolytic treatment.

### Medications

Parenteral anticoagulation for stable patients remains the mainstay of therapy in ED. Low-Molecular-Weight Heparins (LMWHs) and fondaparinux are preferred over unfractionated heparin (UFH) because of lower major bleeding and heparin-induced thrombocytopenia risk.

#### • LMWHs

- Enoxaparin: 1.0 mg/kg, every 12 hours, SC
- Tinzaparin: 175 U/kg, once daily, SC
- Dose reduction is required in renal impairment.

#### • Fondaparinux



## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

### Pediatric considerations

The pediatric pulmonary embolism is relatively rare, but widespread use of CTPA showed that it is more frequent than previously thought.

Up to 30% of adult patients have no identifiable risk factors. Unlike adults, 96-98% of pediatric patients have identifiable risk factors, 88% have two or more. Infants and neonates bear the highest risk. In all age groups, a central venous catheter is the most common risk factor. Other common risk factors include dehydration, septicemia, peripartum asphyxia in neonates. Malignancy, lupus erythematosus, renal disease, congenital thrombophilia, surgery and major trauma are common predisposing factors in older children. Overall, immobilization, hypercoagulability, central venous catheter, excess estrogen state, and concurrent deep venous thrombosis are

- 7.5 mg, body weight 50-100 kg,
- 5 mg, body weight <50 kg
- 10 mg, if body weight >100 kg, once daily, SC)
- Contraindicated, if creatinine clearance <30 mL/min
- Dose reduction by 50%, creatinine clearance is 30-50 mL/min.
- **UFH**
  - 80 Units/kg IV bolus, then 18 Units/kg/h continues IV infusion.
  - Recommended, if the patient is
    - a candidate for thrombolytic treatment
    - severely obese
  - Recommended, if creatinine clearance <30 mL/min
  - Advantages: The ease of monitoring and reversal of effects by protamine.

Thrombolytic treatment must be reserved for unstable patients. Streptokinase, urokinase and recombinant tissue plasminogen activator (rTPA) are approved thrombolytic agents for PE. As a general rule, LMWHs, fondaparinux and UFH infusion must be stopped during thrombolytic therapy. Currently, recombinant tissue plasminogen activator (rtPA) is the most widely used agent and its dose is 100 mg over 2 hours OR, 0.6 mg/kg over 15 minutes, the maximum dose of 50 mg.

Vitamin K antagonists (VKAs, e.g., warfarin) and new oral anticoagulants (NOACs, e.g., dabigatran, rivaroxaban) should be started in the inpatient setting after initial therapy.

Surgical embolectomy, percutaneous catheter-directed treatment, and venous filters are rarely applied after admission to ICU.



associated with pediatric PE. Deep venous thrombosis in children is predominantly associated with upper extremity and central venous catheter rather than lower extremity as in adults.

Pleuritic chest pain (84%), hemoptysis and shortness of breath are the main symptoms. D-dimer and prediction rules are not studied in children. CTPA remains the primary diagnostic tool in the emergency setting. The segmental arteries are affected 52%. The main or central arteries are affected 6%. Children tend to compensate for relatively large clots well out of their cardiopulmonary reserve.

LMWH is the mainstay of the therapy. Hemodynamically unstable patients should receive thrombolysis. The prognosis is generally good. Shock is the predictor of an adverse outcome.

- Enoxaparin
  - <2 months: 1.5 mg/kg/dose SC, twice a day

- >2 months: 1.0 mg/kg/dose SC, twice a day

## Geriatric considerations

The management and treatment do not change in geriatric patients. However, the EP should consider a few issues. The incidence of PE increases with age. Atypical presentations are common; comorbid illnesses and dementia obscure the diagnosis. The treatment does not change, but complications of anticoagulation occur more frequently. The EP must adjust dose according to comorbid situations like renal dysfunction or cachexia.

## Pregnant patient considerations

PE and pregnancy form an ominous couple for apparent reasons. Pregnant and postpartum women are susceptible to PE. Peak times are the third trimester and the first 4 weeks following the labor, particularly after cesarean section. Moreover, breathlessness is a common complaint during pregnancy. The potential harm to fetus and woman breast

from ionizing radiation, the fear of missing a life-threatening diagnosis and the need for quick decisions harden the management of a pregnant woman with suspected PE. A clinical pathway is recommended at this [link](#) as a part of “Emergency Evaluation For Pulmonary Embolism, Part 2: Diagnostic Approach.

The first step is bilateral lower extremity venous ultrasound. If the ultrasound is positive, the treatment starts without further investigation. If the ultrasound is negative, the EP must assess the pretest probability (PTP). The trimester, physician’s gestalt or clinical prediction rules are available methods to assess PTP. Note that no prediction rule is validated in pregnant. In the non-high risk group, PERC negative patients are further stratified with D-dimer. If D-dimer is under cutoff values according to trimesters, PE can be excluded to a reasonable degree of medical certainty. High risk, PERC positive or D-dimer positive patients should undergo imaging. On the imaging

branch, shared decision-making should be pursued between CTPA and ventilation-perfusion scan.

LMWH is safe during pregnancy [Pregnancy Category (PC) B] and lactation and so is standard treatment and is preferred over heparin (PC C). Fondaparinux (PC B) is not recommended due to lack of data. VKAs (PC X) and new oral coagulants are contraindicated in pregnancy. Pregnancy does not alter the dosage.

## Disposition Decisions

### Admission Criteria

- All high-risk patients, including those in shock, who are hypotensive, post-CPR, intubated, or who have received thrombolytic treatment must be admitted to ICU.
- Intermediate-high risk patients should be observed in monitored beds and possibly in ICU
- Intermediate-low risk and low-risk patients should be admitted to the ward.

### Discharge Criteria

- A very selected group of low-risk patients may be treated as outpatients. A proposed algorithm for outpatient management of PE is available at this link
- Several discharge instructions are available online (I and II).

## Referral

- Patients must be referred to respiratory or internal medicine.

## Pearls And Pitfalls

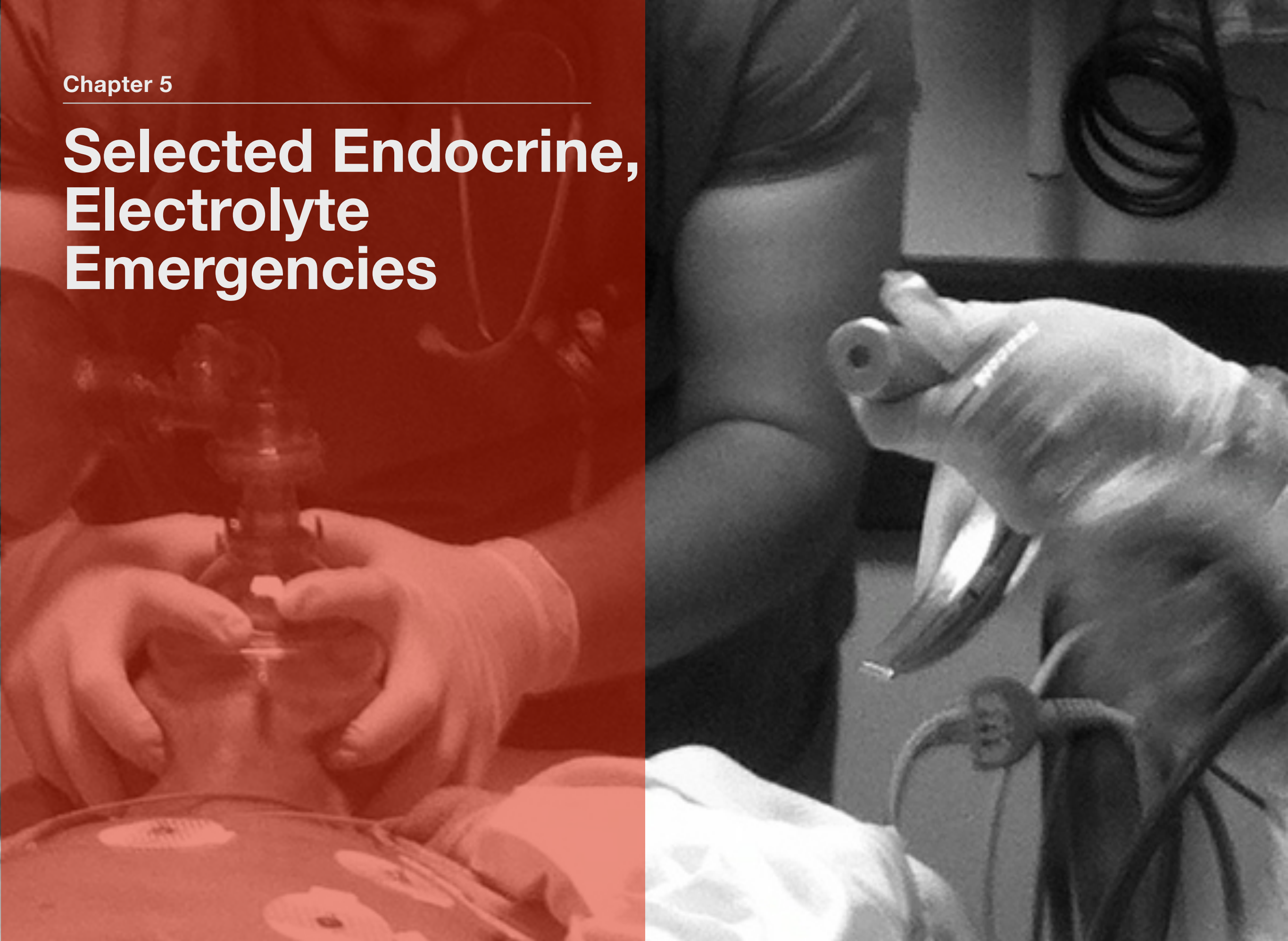
- Use validated clinical prediction rules to estimate pretest probability in patients with suspected PE.
- Do not proceed to D-dimer measurements or imaging studies in patients with a low PTP and negative Pulmonary Embolism Rule-Out Criteria.
- A high sensitivity D-dimer is the initial test in patients with intermediate PTP or low PTP but a positive Pulmonary Embolism Rule-Out Criteria. Imaging studies are not the initial test in patients with low or intermediate PTP.
- Use age-adjusted D-dimer thresholds in patients older than 50.
- CTPA is the initial test in patients with high PTP. Ventilation-perfusion scans are alternative if CTPA is contraindicated or unavailable. D-dimer cannot exclude PE in patients with high PTP.

**References and Further Reading**, click [here](#)

## Chapter 5

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# Selected Endocrine, Electrolyte Emergencies



# Acid-Base Disturbance

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by Lamiess Osman, Qais Abuagla

## Case Presentation

*A 15-year-old female presented with dyspnea, polyuria, and polydipsia for the last 3 days. She was slightly lethargic with dry oral mucosa. Vitals were BP 92/45mmHg, RR 27/bpm, HR 119/bpm, Temp 37°C, SpO2 99% on INO2 1L/min. Physical examination revealed normal findings except there was a mild abdominal tenderness without guarding.*

A bedside arterial blood gas revealed the following:

- pH: 7.19
- PaO2: 105mmHg
- PaCO2: 19mmHg
- HCO3: 7mmol/L
- Na: 124mmol/L
- K: 3.4mmol/L



- Cl: 91mmol/L
- Gluc: 310 mg/dL
- BUN: 13mmol/L

*The patient was put under close monitoring, and intravenous fluids were initiated. Urine dipstick showed glucose 4+ with ketones. A diagnosis of diabetic ketoacidosis was made. The arterial blood gas (ABG) was evaluated at the end of the chapter for this case.*

## Introduction

### Definitions

**Acid:** a substance that is capable of donating a hydrogen ion to another substance

**Base:** the substance that is capable of receiving a hydrogen ion.

**Acidemia** is serum pH < 7.35.

**Alkalemia** is serum pH > 7.45.

**Acidosis** refers to physiologic processes that cause acid accumulation or alkali loss.

**Alkalosis** refers to physiologic processes that cause alkali accumulation or acid loss.

### Defense Mechanisms

The body has three defense mechanisms to maintain normal pH:

1. The buffering system.
2. The respiratory system.
3. The renal system.

Among the three systems, the **buffering** is the fastest means of preventing disturbances in pH. It was likened to a "sponge" that soaks up excessive hydrogen ions and releases them when there's a deficient concentration. Several buffering agents bind



hydrogen ions reversibly. These include bicarbonate, ammonia, hemoglobin and plasma proteins.

The **respiratory** system is a key player in acid-base regulation, albeit acting slightly slower than the buffer system. Central and peripheral chemoreceptors, when stimulated, increase the rate and depth of respiration when carbon dioxide (CO<sub>2</sub>) or hydrogen ions levels are elevated. This increases the rate of CO<sub>2</sub> elimination from the lungs, resulting in less CO<sub>2</sub> available to form carbonic acid.

The **renal** system is a much slower process for dealing with hydrogen ion concentration change, taking hours to days. Therefore, it is more important for the long-term maintenance of acid-base balance. The kidneys can reabsorb bicarbonate and excrete hydrogen ions.

In clinical practice, the recognition of one acid-base disorder must prompt the search for other concurrent disorders. Due diligence must be exercised to complete the evaluation of the arterial

blood gas (ABG) and manage it in the clinical context.

### Critical Bedside Actions and General Approach

The emergency department is where you will first encounter sick patients having critical situations including acid-base disorders. Therefore, you must have the ability to diagnose and manage acid-base derangement. However, every critically ill patient should be evaluated for airway, breathing and circulation in the initial assessment phase and necessary resuscitation effort should be applied. Many of the patients with acid-base problem require cardiac monitorization, IV lines, supplemental fluid and oxygen during the initial evaluation phase.

An acid-base derangement may contribute to the patient symptoms and in some cases signifies an immediate life threat.

The body produces acid as a byproduct of basal metabolism. Respiratory acids

are made up of carbon dioxide; as an end product of the metabolism of carbohydrates and fats. Although CO<sub>2</sub> is not an acid in itself, when combined with water it forms carbonic acid, which is capable of dissociating to hydrogen ions and bicarbonate ions. Lungs can excrete CO<sub>2</sub> it is known as volatile acids. However, many acids are referred to as fixed acids such as ketoacidosis and lactic acid, and they depend on the kidneys for their excretion.

The body concentration of hydrogen ions must be maintained within a strict range for optimal cellular function, and even a small deviation can significantly affect the patient. This underscores the importance of being able to evaluate a blood gas for an acid-base disorder. Essentially, this is accomplished by these laboratory values: pH, PaCO<sub>2</sub>, and serum HCO<sub>3</sub><sup>-</sup>. Na and Cl are also required in the anion gap analysis.

## 5 Simple Steps to Solve an Acid-Base Problem

### Illustration 5.1

**iEM** INTERNATIONAL EMERGENCY MEDICINE EDUCATION PROJECT

### 5 SIMPLE STEPS TO SOLVE ACID-BASE PROBLEM

- 1 ORDER LABS**  
VBG/ABG LACTATE CHEMISTRY
- 2 DETERMINE THE PH**  
ACIDEMIA pH < 7.35  
ALKALEMIA pH > 7.45
- 3 DETERMINE THE PRIMARY PROCESS**

METABOLIC ACIDOSIS	METABOLIC ALKALOSIS	RESPIRATORY ACIDOSIS	RESPIRATORY ALKALOSIS
HCO <sub>3</sub> <sup>-</sup> < 22 mEq/L	HCO <sub>3</sub> <sup>-</sup> > 26 mEq/L	PaCO <sub>2</sub> > 45 mmHg	PaCO <sub>2</sub> < 35 mmHg
- 4 PRESENCE OF COMPENSATION VS. CONCOMITANT PRIMARY PROCESS**

METABOLIC ACIDOSIS	METABOLIC ALKALOSIS	RESPIRATORY ACIDOSIS	RESPIRATORY ALKALOSIS
Winter's formula $pCO_2 = 1.5[HCO_3^-] + 8 (\pm 2)$ or The last 2 digits of the pH	$pCO_2 = 0.7[HCO_3^-] - 2 (\pm 2)$ "The maximum respiratory compensation is around PaCO <sub>2</sub> of 55mmHg, as hypoxia from the 'compensating' alveolar hypoventilation will stimulate respiration."	In acute cases for every 10mmHg increase in [pCO <sub>2</sub> ], [HCO <sub>3</sub> <sup>-</sup> ] increases by 1mEq/L.  In chronic cases for every 10mmHg increase in [pCO <sub>2</sub> ], [HCO <sub>3</sub> <sup>-</sup> ] increases by 3.5 mEq/L.	In acute cases for every 10mmHg decrease in [pCO <sub>2</sub> ], [HCO <sub>3</sub> <sup>-</sup> ] decrease by 2mEq/L.  In chronic cases for every 10mmHg decrease in [pCO <sub>2</sub> ], [HCO <sub>3</sub> <sup>-</sup> ] decrease by 5 mEq/L.
- 5 LOOK FOR THE CAUSE OF EACH CATEGORY**

METABOLIC ACIDOSIS	METABOLIC ALKALOSIS	RESPIRATORY ACIDOSIS	RESPIRATORY ALKALOSIS
Calculate Anion Gap $Na - (Cl + HCO_3)$ Normal anion gap is 8-16  High AG MA (AG >16) MUD PILES Methanol Uremia DKA Paraldehyde Isoniazid Lactic Acidosis EtOH/Ethylene glycol Salicylates  Normal AG MA (AG <16)  HARDUPS Hyperalimentation/Hypotension Acetazolamide Renal tubular acidosis Diarrhea Uretero-Pelvic shunt Post hypocapnia Spironolactone	Obtain a urine Chloride level  CLEVER PD Contraction (due to blood loss) Uricemia Endocrine (Conn's/ Cushing's/ Batters') Vomiting/nasogastric suction Excess Alkali Refeeding Alkalosis Post-hypercapnia Diuretics  (* associated with high urine Cl level)	From a pathophysiological perspective, the two broad categories are V/Q mismatch with/out increased CO <sub>2</sub> production, and alveolar hypoventilation. Causes can also be classified according to its acuity.  Acute causes: CNS depression (cerebrovascular accident/drugs) Airway obstruction Pneumonia Pulmonary edema Hemo/pneumothorax Myopathy Chronic cause: Chronic obstructive pulmonary disease (COPD) Restrictive lung disease	This is secondary to excessive ventilation, i.e., excessive respiratory rate and/or depth from the following causes:  CNS disease Hypoxia Anxiety Mechanical ventilation Progesterone Salicylates Sepsis  REFERENCES wikim.org lifeinthefastlane.com iem-student.org iem_student

## Metabolic Acidosis

- Calculate the Anion Gap.  $AG = Na - (Cl + HCO_3)$
- Normal anion gap is 8-16.
- Causes of high anion gap metabolic acidosis (i.e.,  $AG > 16$ ) can be remembered using mnemonic: "MUD PILES."

• Methanol

• Uremia

• DKA

• Paraldehyde

• Isoniazid

• Lactic Acidosis

• EtOH/Ethylene glycol

• Salicylates

- Causes of a normal anion gap metabolic acidosis (i.e.,  $AG < 16$ ) can be remembered using the mnemonic: "HARDUPS"

• Hyperalimentation/  
Hypoaldosteronism

• Acetazolamide

• Renal tubular acidosis

• Diarrhea

• Uretero-Pelvic shunt

• Post hypocapnia

• Spironolactone

## Respiratory Acidosis

From a pathophysiological perspective, the two broad categories are V/Q mismatch with/out increased CO<sub>2</sub> production, and alveolar hypoventilation due to either central causes or chest wall-neuromuscular disorders. Causes can also be classified according to its acuity.

• Acute causes:

• CNS depression (cerebrovascular accident/drugs)

• Airway obstruction

- Pneumonia
- Pulmonary edema
- Hemo/pneumothorax
- Myopathy
- Chronic cause:
  - Chronic obstructive pulmonary disease (COPD)
  - Restrictive lung disease

## Metabolic Alkalosis

Obtain a urine Chloride level and remember the following mnemonic: **“CLEVER PD”**

- Contraction (due to blood loss)
- Licorice \*
- Endocrine (Conn’s/ Cushing’s/ Batter’s)\*
- Vomiting/nasogastric suction
- Excess Alkali\*
- Refeeding Alkalosis\*

- Post-hypercapnia
  - Diuretics\*
- (\* associated with high urine Cl level)

## Respiratory Alkalosis

This is secondary to excessive ventilation, i.e., excessive respiratory rate and/or depth from the following causes:

- CNS disease
- Hypoxia
- Anxiety
- Mechanical ventilation
- Progesterone
- Salicylates
- Sepsis

## Looking Back To Our Case

### Step 1: Interpretation of pH

- The pH <7.35 indicating acidosis

**Step 2:** Evaluate the primary process that accounts for the deranged pH.

- The low  $\text{HCO}_3^-$  with a low  $\text{PaCO}_2$  indicates that the main primary disorder is metabolic acidosis.

**Step 3:** Evaluate for appropriate, over or under compensation: indicating the concurrent primary acid-base disorder in the compensating system.

- Using the Winter’s formula  $\{\text{expected } \text{PaCO}_2 = 1.5[\text{HCO}_3^-] + 8 (\pm 2)\}$ , the expected  $\text{PaCO}_2$  is 18.5. The patient’s actual  $\text{PaCO}_2$  (19mmHg) lies within this range. This means that the respiratory compensation is appropriate and there was no concurrent respiratory acid-based disorder.

**Step 4:** Calculate the Anion Gap, Anion Gap and delta  $\text{HCO}_3^-$

$\text{AG} = \text{Na} - (\text{HCO}_3^- + \text{Cl}) \pm 4$ . The patient’s AG is  $26 \pm 4$  {i.e.  $124 - (7 + 91) \pm 4$ }.

$\text{AG} = \text{AG} - 12$ . The patient’s AG is  $14 \pm 4$  {i.e.  $26 - 12 (\pm 4)$ }.

Delta  $\text{HCO}_3^- = 24 - \text{HCO}_3^-$ . The patient's delta  $\text{HCO}_3^-$  is 17 {i.e.  $24 - 7$ }

Since this drop in  $\text{HCO}_3^-$  (17) lies within the increased in AG ( $14 \pm 4$ ), there is no second metabolic disorder.

In conclusion, the patient's ABG is a pure High Anion Gap Metabolic Acidosis. As a Step 5, look for the causes of defined acid/base situation. In this case, use MUD PILES mnemonic to find specific problem.

**References and Further Reading**, click [here](#)

# Hyperglycemia

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by Toh Hong Chuen

## Case Presentation

*A 58-year-old lady presented with right foot pain for 3 days, associated with high fever, lethargy, polyuria, and polydipsia. At triage, air hunger was noted. Her vital signs were: BP 82/46 mmHg, PR 131/min, RR 28/min, T 38.7 and SpO2 98%. She was brought to the resuscitation room for further management.*

*Clinically, she was dehydrated and confused with GCS E4V4M6. Her neck was supple, and lungs were clear. Crepitus was noted on the dorsum of the right foot. Point of care blood tests showed: capillary glucose 40 mmol/L, capillary ketone 7.2 mmol/L, pH 7.22, HCO3 8 mmol/L, pCO2 20 mmHg, Na 130 mmol/L, Cl 95 mmol/L, K 5.5 mmol/L and lactate 6.9 mmol/L.*



Audio is available [here](#)



*A diagnosis of septic shock secondary to gas gangrene complicated by diabetic ketoacidosis was made. She was aggressively resuscitated with fluid and started on IV insulin infusion. Potassium replacement was withheld as K was elevated. Urinary catheterization was performed for strict input-output monitoring. Broad-spectrum antibiotics and intramuscular tetanus toxoid were given. X-ray (Image 5.1) of right foot confirmed subcutaneous air.*

Image 5.1



*The patient was sent directly to the theatre and underwent extensive debridement for the gas gangrene. She had an uneventful recovery and was discharged 1 week later.*

## Critical Bedside Actions and General Approach

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are potentially life-threatening diabetic emergencies. In acutely ill patients with hyperglycemia, blood and urine tests must be performed, preferably at the point of care, to evaluate for the presence of DKA or HHS (Table 5.1).

Key management principles for DKA and HHS include initiation of IV fluid therapy and IV insulin infusion while ensuring normokalemia and avoiding hypoglycemia. The precipitating cause should be identified and treated. Patient's vital signs, mental status and biochemical response to therapy (glucose, ketones, Na and K) and input-output must be closely monitored. As DKA and HHS resolve, overlap with s/c insulin prior to stopping the insulin infusion. See [appendix 1](#) for management details.

## Differential Diagnoses

**Table 5.1** Diagnostic Criteria For DKA And HHS

TERIA	DKA MILD	DKA MODERATE	DKA SEVERE	HHS
Serum glucose (mmol/L)	≥14	≥14	≥14	≥33
pH	7.25-7.30	7.00-7.24	<7.00	>7.3
HCO <sub>3</sub> (mmol/L)	15-18	10-14	<10	>18
Anion gap (mmol/L)	>10	>12	>12	Variable
Serum ketone (mmol/L)	≥0.6	≥0.6	≥0.6	Small/none
Urine ketone	Positive	Positive	Positive	Small/none
Effective serum osmolality (mOsm/kg)	Variable	Variable	Variable	>320

Adapted from Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. *Diabetes Care*. 2009;32(7):1335-1343. doi:10.2337/dc09-9032. Please read the article for more information.

**D K A   a n d   H H S   a r e distinguished as follow:**

Hallmarks of DKA are anion-gap acidosis, ketosis, and hyperglycemia.

- Usually associated with type 1 diabetes; can occur in type 2 diabetes during a serious illness.
- A short duration of symptoms, usually <1-2 days.
- Up to 10% of DKA are “euglycaemic” (glucose <14). They can be seen in pregnant patients, those with restricted food intake, or had initiated insulin therapy (though insufficient) prior to presentation.

Hallmarks of HHS are profound hyperosmolality, hyperglycemia, and dehydration.

- Associated with type 2 diabetes; can occur with type 1 as a simultaneous occurrence with DKA.
- Like DKA, the circulating amount of insulin is inadequate to prevent hyperglycemia. Unlike DKA, this amount is sufficient to prevent lipolysis and ketoacidosis.
- Without significant ketoacidosis, HHS develops slowly and subtly over several days, contributing to more severe water deficit at around 7-12 L, compared to 4-6 L in DKA.

- The older terms “hyperglycemic hyperosmolar nonketotic coma” (HHNK) and “hyperglycemic hyperosmolar nonketotic state” (HHNS) should not be used. Patients often present without coma, and ketonemia may be found in some.

Up to 1/3 of patients have an overlap of DKA and HHS.

Any significant stress can precipitate DKA/HHS, remembered as “7 I’s have bled!”.

- Iatrogenic (drug interaction, e.g. steroids)
- Idiopathic (new onset DM)
- Illegal (substance abuse)
- Infarction (e.g. AMI, stroke, bowel ischemia)
- Infection (e.g. pneumonia, UTI, cellulitis)
- Infraction (i.e. noncompliance)
- IUP (i.e. pregnancy)

- Bleeding GI

## History and Physical Examination Hints

1. DKA and HHS can present similarly with malaise, anorexia, thirst, polyuria, and polydipsia. In addition, they can be triggered by similar precipitants. Differences include a longer presentation and more severe dehydration in HHS.

### 2. Neurological symptoms

- HHS patients often have some degree of altered mental state or other neurological disturbances. These are related to the severity and rate of development of hyperosmolality.
- Conversely, since hyperosmolality is absent or insignificant, DKA patients have normal neurological status. Only severe DKA presents with coma.

### 3. Respiratory symptoms

- DKA patients often present with air hunger and Kussmaul’s breathing secondary to acidosis, unlike HHS.

### 4. Abdominal symptoms

- In DKA, nausea, vomiting and abdominal pain are associated with the severity of ketoacidosis.
- Conversely, an acute abdominal process such as pancreatitis can precipitate DKA. Search for intraabdominal precipitants if abdominal pain when DKA is mild, persistent pain despite improvement of acidosis or signs of peritonism.
- HHS (no significant ketoacidosis) is not associated with abdominal pain. Evaluate for an abdominal precipitant if there are abdominal symptoms.

5. Physical findings may be unreliable for estimating the degree of dehydration, particularly in children

- In DKA, patients may appear more dehydrated from the drying of oral mucosa due to Kussmaul's respiration.
- Hyperosmolality in HHS may "preserve" intravascular volume (even though it leads to urinary losses) and mask signs of volume depletion until hemodynamic deterioration suddenly occur.

## Emergency Diagnostic Tests and Interpretation

### High Anion Gap Metabolic Acidosis (HAGMA)

- The most important feature of DKA.
- Anion gap =  $\text{Na} - (\text{Cl} + \text{HCO}_3)$ . Use measured sodium in the calculation of anion gap.
- pH in venous blood gas is sufficient as it correlates with arterial pH. Perform arterial blood gas only if concomitant respiratory failure is suspected.

- Note that the severity of metabolic acidosis can be masked by metabolic alkalosis from vomiting.
- Interestingly, most patients change from HAGMA to NAGMA while recovering from treatment. This is due to urinary loss of ketones earlier during osmotic diuresis. Ketones can be metabolized to bicarbonate when adequate insulin is provided; hence the loss of urinary ketones is equivalent to losing bicarbonate, resulting in NAGMA.

### Ketosis

- The small amount of ketones is normally present ( $<0.6$  mmol/L) acting as an alternative energy source if glucose is not available.
- In DKA, relative or absolute insulin deficiency and the surge of counterregulatory hormones (especially glucagon and catecholamines) cause unrestrained ketogenesis. All three ketones, acetone (Ac), acetoacetate (AAc) and beta-hydroxybutyrate (BHB) are elevated.

• AAc and BHB fully dissociate in physiological pH and contribute to HAGMA. Ac, which does not dissociate, does not.

- BHB is the most abundant ketone in DKA, with a ratio of 10:1 compared to AAc, and Ac is least abundant.
- Insulin reduces overall ketone level but also converts BHB to AAc. As nitroprusside-based urine test detects only Ac and AAc, urinary ketones may not improve or paradoxically worsen with treatment. Therefore, serum BHB should be used to monitor resolution of ketosis.
- Note that blood test for ketones can be falsely positive in a patient taking sulfhydryl drugs.

### Serum osmolality

- The effective serum osmolality should be used in the diagnosis of HHS, not measured osmolality. Measured Na should be used to determine the osmolality.

- Effective serum osmolality =  $2 \times \text{Na (mmol/L)} + \text{Glucose (mmol/L)}$ .
- Urea travels freely across a cell membrane and does not contribute to osmolality in vivo.

### Serum potassium

- Check K before and after starting insulin.
- Total body potassium depletion occurs through urinary (and occasionally gastrointestinal) loss. Serum K, however, may be normal or paradoxically elevated due to transcellular shift (acidosis, insulin deficiency), volume contraction and reduced renal function.
- Hypokalemia at presentation signifies profound K loss. This generally worsens with treatment and may precipitate threatening arrhythmia and profound respiratory muscle weakness.

### Serum sodium

- As hyperglycemia draws fluid into the intravascular space, most patients have

low normal or mild hyponatremia (dilutional). Hypernatraemia, therefore, signifies severe dehydration.

- Use the measured Na when calculating the anion gap and serum osmolality.
- Corrected Na = serum Na +  $2 \left[ \frac{\text{serum glucose} - 5.5}{5.5} \right]$
- After the initial fluid challenge with 0.9% NaCl, use corrected sodium to decide on the choice of saline for infusion.

### Others

- Leukocytosis is present due to the elevated levels of stress hormones. Up to 15K may be expected for DKA.
- Serum amylase, lipase, hepatic enzymes, creatinine kinase and CRP, can be mildly elevated. These are nonspecific findings.

## Emergency Treatment Options

### Fluids

- Patients with DKA and HHS are invariably volume depleted. Start IV 0.9% NaCl at 10-20 mmol/kg/hr during the first hour.
- After BP and perfusion normalizes, continue infusion at a rate of 250-500 ml/hr with 0.45% NaCl if the calculated Na is normal or high; or 0.9% NaCl if the calculated Na is low
- The total body fluid deficit should be slowly corrected over 24 hrs.

### IV insulin

- Mainstay treatment of DKA and HHS.
- Before initiating IV insulin,
  - Initiate fluid replacement. With insulin, glucose is taken up by cells, drawing fluid out of intravascular space and can cause hypotension.



- Correct hypokalemia, if present, with IV KCl at 20-30 mmol/hr until  $K > 3.3$  mmol/L.
- Give as a continuous infusion of 0.1 U/kg/hr, and not as a bolus as this may cause severe hypokalemia and may risk hypoglycemia.
- Resolution of DKA as indicated by serum glucose  $< 11$  mmol/L plus any 2 of the following:
  - pH  $> 7.3$ ,
  - $HCO_3^- > 18$  mmol/L
  - Anion Gap  $\leq 12$  mmol/L
- As the resolution of ketoacidosis in DKA often lags behind hyperglycemia (mean duration of 12 hours versus 6 hours respectively), IV insulin should be continued with dextrose replacement to clear the ketones. When capillary glucose reaches 11 mmol/L:
  - Reduce IV insulin rate to 0.02-0.5 U/kg/hr

- Add D5% to replacement fluid, keep serum glucose between 8-11 mmol/L until ketoacidosis resolves.
- Once resolved and the patient is able to take orally, start subcutaneous rapid-acting insulin at 0.1 U/kg around 30-60 min before stopping the insulin infusion, given the delayed onset of the s/c preparation, to prevent rebound hyperglycemia.
- Similarly in HHS, when capillary glucose reaches 16 mmol/L:
  - Reduce IV insulin rate to 0.02-0.5 U/kg/hr, keep serum glucose between 11-16 mmol/L.
  - IV insulin can be stopped once the patient is alert, taking orally and started on their oral hypoglycemic or subcutaneous insulin.

## Potassium

- Correct hypokalemia if present as above.

- Add 20-30 mmol of K in each liter of fluid to maintain normokalemia.
- Withhold K if elevated above the upper limit (or  $> 5.2$  mmol/L).
- Check K 2 hourly after initiating fluid and insulin therapy

## Phosphate

- DKA patients have total body phosphate though serum levels may be normal or elevated. Treatment with insulin drives phosphate intracellularly and worsens hypophosphatemia.
  - However, routine phosphate replacement is not indicated as no studies demonstrated benefit. Treatment could also precipitate hypocalcemia.
- Consider phosphate replacement (IV K<sub>2</sub>PO<sub>4</sub> at 4.5 mmol/hr) in DKA if:
  - Cardiac dysfunction
  - Respiratory depression
  - Anemia

- Severe hypophosphatemia (<1 mg/dL).
- Unlike DKA, there is no evidence for replacement of phosphate in HHS.

## Sodium Bicarbonate

- NaHCO<sub>3</sub> should not be given in DKA as it is Independent risk factor for cerebral edema, and can worsen hypokalemia, create excessive sodium load, induce paradoxical CSF acidosis or even impairing release of O<sub>2</sub> to tissues via shifting the oxygen-hemoglobin curve to left.
- Consider only if the patient is severely acidotic (pH <6.9).

## Pediatric, Geriatric, Pregnant Patient and Other Considerations

### Pregnancy (DKA)

- Low maternal mortality (<1%), but perinatal mortality can be as high as 35%.

- Presents similarly to non-pregnant women, but 1/3 of patients may have euglycaemic DKA.
- Treated similarly, but include fetal monitoring.

### Pediatric (DKA)

- As the clinical judgment of dehydration is unreliable, set maintenance fluids based on of 6% total water deficit.
- The most feared complication is cerebral edema
- Occurs in 1% of DKA episodes, but high mortality of 40-90%.
- Major risk factors include treatment with sodium bicarbonate and severe hypocapnia and high urea at presentation.
- Typically presents 4-12 hours after initiating treatment, but can be delayed up to 48 hours. Once recognized: elevate the head of the bed and reduce IV fluids by 1/3. If BP is normal, start IV mannitol (0.5-1 g/kg over 20 min). If

patient is hypotensive, use IV 3% NaCl (5-10 ml/kg over 30 min).

## Geriatric (HHS)

- Prone to develop HHS due to altered thirst mechanism, restricted access to water and decreased pancreatic reserve. Always check blood sugar in patients, especially the elderly, with an altered mental state.
- Managed similarly to other population.

## Disposition Decisions

All patients with DKA or HHS should be admitted. They should be sent to the ICU or high dependency unit if there were severe symptoms, refractory hypotension, refractory oliguria or persistent mental state changes.

**References and Further Reading**, click [here](#)

# Hypernatremia

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by Vigor Arva, Gregor Prosen

## Case Presentation

*A 79-year-old man was brought to the emergency department (ED) by his wife. She complained that the patient had general weakness and was feeling 'unwell' for the last two days. He had a history of dementia, diabetes, renal failure, and hypertension. He was on diabetic and antihypertensive medication.*

*On examination, his vital signs were as follow HR 115/min, BP 135/90 mmHg, RR 17/min, and afebrile with normal oxygen saturation. He was confused and disoriented, but there was no other deficits or localizing signs on neurological exam. He was clinically dehydrated with dry oral mucosa. Lab results showed a serum sodium concentration of 160 mEq/L, with elevated glucose, creatinine, urea, and osmolality. Point of care ultrasound demonstrated a small and almost totally-collapsed inferior vena cava. Upon further history taking, the*



Audio is available [here](#)

*patient's wife reported that he had not been drinking much for the last few days, even though he did not complain about thirst.*

## **Critical Bedside      Actions and General Approach**

Hypernatremia should be excluded in any patient who presents with altered mental status, particularly, the very young or old and those with abnormal basal cognition.

Once diagnosed, the next step is to assess for volume status and acuity of symptoms, as they influence treatment plan. It is considered acute if symptoms develop within 48hrs, and chronic if it is longer. Then, the cause should be determined and treated.

## **Differential Diagnoses**

Hypernatremia usually results from relative water losses, and rarely secondary to sodium overload. The causes can be categorized into the following three groups:

A. Hypernatremia with hypovolemia: Often found with dehydration and fluid losses such as heatstroke, excessive sweating, burns, and gastrointestinal losses with diarrhea and vomiting

B. Hypernatremia with euvolemia: Occurs mostly as a result of ADH deficiency, diabetes insipidus, reset of osmostat, hypothalamic dysfunction, primary hypodipsia, renal diseases, drugs and alcohol (e.g., lithium, amphotericin, phenytoin, aminoglycosides)

C. Hypernatremia with hypervolemia: Can occur because of iatrogenic causes infusion of hypertonic sodium solutions (3%

saline, or large volumes of sodium bicarbonate), ingestion of salt water or large amounts of salt, Cushing's and Conn's syndrome

Diabetes insipidus (DI) refers to an absolute or relative antidiuretic hormone (ADH) deficiency. Absolute ADH deficiency occurs in the setting of inadequate ADH secretion and is called central DI. Relative ADH deficiency occurs in lack of renal response to ADH and is called nephrogenic DI.

Malignant diseases, trauma or surgery on the pituitary, infiltrative diseases, familial diseases or idiopathic conditions may cause central DI. Chronic renal insufficiency, tubulointerstitial diseases, polycystic kidney disease, hypercalcemia, hypokalemia, lithium toxicity, or familial diseases may cause nephrogenic DI.

Symptomatic hypernatraemia (e.g., polyuria and polydipsia, lethargy and weakness) with an inappropriately low urine osmolality ( $<300$  mOsm/kg) should suggest DI in the ED. The formal diagnosis requires a water deprivation test, which is often not performed in the ED given the long duration required.

Central and nephrogenic DI are further distinguished by the response to desmopressin (synthetic ADH). With desmopressin, urine osmolality will rise to more than 800 mOsm/kg in patients with central DI, while this rise is absent in nephrogenic DI.

Psychogenic polydipsia can be distinguished from DI by water restriction. Following water restriction, urine osmolality will rise in psychogenic polydipsia and remain unchanged in DI.

## History and Hints

The signs and symptoms of hypernatremia are nonspecific including lethargy, irritability, restlessness, hyperactive reflexes, and increased muscle tone. Severe symptoms usually occur after the serum Na has risen acutely above 158 mEq/L, and may include seizures, coma or even death.

Rapid and severe hypernatremia developing over minutes and hours can result in brain hemorrhage, due to the accompanying rapid decrease in brain volume causing ruptures of cerebral veins. It is also associated with hypocalcemia, for unclear reasons.

If the hypernatremia is chronic, the brain can adapt by generating intracellular osmogenic compounds, or idiogenic osmoles, which increases the osmolality in the cells and thus maintaining brain volume by resisting shrinkage.

Patients should be asked about their fluid and salt intake, urine output, and concurrent medical and medication history. The patient's caregiver should be interviewed, especially if the patient is mentally altered, to see if there are mental or behavioral changes (e.g., excessive water intake).

Patients should be examined for their volume status by checking skin turgor, capillary refill, looking for edema and raised jugular venous pressure, measuring heart rate, blood pressure and looking for a postural drop, mental and neurological status.



## Emergency Diagnostic Tests and Interpretation

Serum sodium level (Normal reference: 135-145 mEq/L) and serum osmolality (Normal reference: 275-295 mOsm/L) should be checked. Both can be done at the bedside. Other electrolytes (mainly potassium and calcium) should be checked. Patients urine output and urine osmolality should be measured.

Total body water (TBW) deficit can be calculated as a function of sodium concentration:

$$TBW \text{ deficit} = TBW \times [(serum \text{ sodium}/140) - 1]$$

Average total body water is obtained by multiplying the patient's body mass by 0.6. However, this holds mostly for children and adult men. A multiplier of 0.5 for elderly men and adult women and 0.45 for elderly women should be preferred.

## Emergency Treatment Options

Following the initial ABC evaluation, hypernatremic patients with hemodynamic instability should be resuscitated with intravenous fluids. The aim at this point is to address the underlying hypovolemia and tissue hypoperfusion. Unless this is corrected, the body's normal response is to increase the sodium concentration even further to maintain intravascular volume, which will worsen the hypernatremia. Fluid resuscitation can be achieved using normal saline (0.9%) or Ringer's lactate.

After stabilizing the patient, proceed to evaluate for and treat the underlying cause of hypernatremia (e.g., fever, new medicine, gastrointestinal disturbances and DI).

In patients with acute hypernatremia (symptoms occurring <48hrs), it is relatively safe to correct sodium at a rate of 1mEq/L/hr, with correction of the total water deficit in 24 hours. This is because the brain did not have enough time to produce idiogenic osmoles, lowering the risk for cerebral edema.

In patients suspected of having chronic hypernatremia, the sodium should be corrected gradually, and no more than 0.5 mEq/hr or 10-12 mEq/day. Rapid overcorrection can result in cerebral edema, especially in children. Oral rehydration with water may be the safest option.

### Sodium concentration in different solutions

- 0.9% NaCl: 154 mmol/L
- Ringer's Lactate: 130 mmol/L
- 0.45% NaCl: 77 mmol/L
- 5% Dextrose in water (D5W): 0 mmol/L

To determine the volume and rate of correction, one can calculate it manually using the formulas given above. There are also several free online calculators available as well, such as the one by [Medcalc](#).

Patients with known central DI should be given desmopressin (DDAVP) which should improve symptoms. Initial doses are 1 – 2 micrograms. The dose should be up-titrated if necessary.

## **Pediatric, Geriatric, Pregnant Patient, and Other Considerations**

Infants can develop hypernatremia if they are not given adequate fluids, or are given hypertonic fluids. Older children can also develop hypernatraemia with volume depletion following after severe diarrhea or vomiting.

Pregnant patients may develop hypernatremia if vomiting secondary to hyperemesis gravidarum is severe. Cases of gestational diabetes insipidus have been documented and should not be confused with polyuria that can normally occur during pregnancy.

Hypernatremia is common in the elderly. The causes are likely multi-factorial and inter-related, including the decrease in thirst sensation, polypharmacy, and pre-existing co-morbidities.

## **Disposition Decisions**

Patients with symptomatic hypernatremia should be admitted for evaluation and treatment, as the free water deficit is generally replaced gradually. Those with severe neurological symptoms will require admission to a closely monitored unit.

Hemodynamically stable and asymptomatic patients with mild hypernatremia from benign causes may be discharged with advice and follow up with the primary physician.

**References and Further Reading**, click [here](#)

# Hyponatremia

---

by Vigor Arva, Gregor Prosen

## Case Presentation

*A 72-year-old man was brought to the emergency department (ED) by his daughter. She reported that he had nausea, vomiting, and confusion and had been unwell for the last few days. He had hypertension and heart failure for the previous ten years and was on ACE-inhibitor, beta-blocker and thiazide diuretic.*

*At triage, the patient's vital signs were usual: BP 110/70 mmHg, HR 95/min, RR 15/min, temperature 36.1°C and SpO2 100% on room air. He appeared lethargic and walked with an unsteady gait. He had no focal neurological deficit. He had a normal skin turgor and no edema. Postural BP revealed mild orthostatic hypotension. The lab results showed a serum sodium concentration of 115 mEq/L.*



Audio is available [here](#)

## Critical Bedside Actions and General Approach

Hyponatremia is the most frequent electrolyte disturbance and refers to when the serum Na is  $<135\text{mEq/L}$ . The management principles are as follow:

1. Assess severity and acuity
2. Determine type (based on plasma osmolality and volume status)
3. Identify and treat the underlying cause
4. Prevent complications

### Hyponatremia can be classified as

- mild ( $125\text{-}134\text{ mEq/L}$ ),
- moderate ( $120\text{-}124\text{ mEq/L}$ )
- severe ( $<120\text{ mEq/L}$ )

The physician should carefully evaluate the underlying cause before attributing the symptoms to hyponatremia alone as mild and moderate hyponatremia are often asymptomatic. Determining the rate of change is important since rapid

changes of sodium concentration usually present more dramatically with more neurologic involvement. The management differs based on the rate of change.

## Differential Diagnoses

The type of hyponatremia has to be determined to narrow down the differential diagnoses. Based on serum osmolality, we distinguish between hypertonic, isotonic, and hypotonic hyponatremia.

**Hypertonic hyponatremia** (osmotic pressure  $> 295\text{ mOsm/L}$ ) occurs when a large concentration of osmotically active substances pull additional water and dilute sodium concentration.

- Hyperglycemia (for each  $1\text{mmol/L}$  rise in blood glucose, the serum sodium decreases by  $0.3\text{mmol/L}$ )
- Administration of osmotic agents, such as mannitol, glycerol, sorbitol, and radiocontrast infusion

**Isotonic hyponatremia** (osmotic pressure  $275\text{-}295\text{ mOsm/L}$ ) (Also known as pseudo-hyponatremia) occurs with high levels of osmotically inactive substances.

- Hyperlipidemia
- Hyperproteinemia (multiple myeloma etc.)

**Hypotonic hyponatremia** (osmotic pressure  $< 275\text{ mOsm/L}$ ) is subdivided based on clinical evaluation of volume status and urine sodium concentration.

### A. Hypovolemic

- Urinary sodium  $> 20\text{mEq/L}$ : renal losses (diuretics, salt-wasting nephropathy, mineralocorticoid deficiency)
- Urinary sodium  $<20\text{mEq/L}$ : extrarenal (hypotonic fluids, GI, and third space loss, sweating in CF patients)

### B. Euvolemic

- Syndrome of inappropriate ADH secretion (SIADH)
- Drugs
- Hypothyroidism
- Pain, stress, nausea, psychosis, water intoxication

### C. Hypervolemic

- Urinary sodium > 20 mEq/L: renal failure
- Urinary sodium < 20 mEq/L: CHF, nephrotic syndrome, liver cirrhosis

If SIADH is suspected, evaluate for its cause. The acronym MADCHOP lists the causes of SIADH:

- Malignancy
- ADH secretion (ectopic)
- Drugs (e.g., SSRIs, ecstasy)
- CNS disease

- Hormone deficiency (hypothyroidism, adrenal insufficiency)

- Others

- Pulmonary

Drugs that commonly cause hyponatremia include ADH, nicotine, MDMA, SSRI, sulfonylureas, opioids, barbiturates, NSAIDs, paracetamol, carbamazepine, phenothiazine, TCA, colchicine, cyclophosphamide, and MAOI.

## History and Physical Examination Hints

The signs and symptoms of hyponatremia are usually vague and nonspecific. Patients may present with nausea, vomiting, anorexia and general malaise. Pay attention to neurologic signs including a headache, confusion, lethargy, seizures, and coma. Hyponatremia can also manifest as muscle cramps, ataxia, hemiparesis, and focal weaknesses. Symptoms may

develop acutely (<24h) or may develop gradually and subtly over days.

Ask patients about their fluid intake (polydipsia) and losses, comorbidities (e.g., heart and renal diseases, malignancies), medications (e.g., thiazide diuretics) and any recent illness (gastrointestinal diseases with vomiting or diarrhea), or use of recreational drugs.

During the physical examination, attention should be focused on neurological examination and volume status. The latter can be assessed by checking skin turgor, capillary refill, and whether pitting edema, raised jugular venous pressure, and orthostatic hypotension are present. The signs of the common causes of hyponatremia, such as cirrhosis and heart failure, should be carefully elicited.

## Emergency Diagnostic Tests and Interpretation

If hyponatremia is suspected, check the serum sodium level (Normal reference: 135-145 mEq/L) and serum osmolality



(Normal reference: 275-295 mOsm/L), preferably at the bedside if available. Serum osmolality can also be calculated by using the following formula:

$$\text{Osmolality (in mmol/L)} = 2 \text{ Na} + \text{Glucose} + \text{Urea}$$

After the type of hyponatremia, Checking the urine sodium and osmolality to evaluate the etiology further. Note to monitor these values after treatment for hyponatremia is initiated.

If pseudohyponatremia is suspected, verify if the serum sodium measure is affected by protein and lipid concentration. If this is the case, add serum protein and lipid levels to exclude pseudohyponatremia.

Exclude other electrolytes abnormalities, especially potassium. These disorders often co-exist in conditions such as adrenal insufficiency.

Point of care ultrasound can be used to evaluate volume status. Nerve sheath diameter can be measured using ocular ultrasound if cerebral edema is suspected.

## Emergency Treatment Options

Initiate therapy with 100 mL of 3% NaCl over 10-15 minutes in patients with severe acute hyponatremia (sodium < 120 mEq/L) and neurological symptoms, such as seizures, confusion or coma. At this point, recheck the serum sodium. Give a second dose of 3% NaCl if hyponatremia is still severe and the patient

still symptomatic. Additional doses are not advised given the risk of overcorrection. If 3% NaCl is not available, consider using NaHCO<sub>3</sub> solution.

If the patient is stable, evaluate the patient's volume status to determine the management strategy.

Hypovolemic patients benefit more from increasing volume rather than sodium correction per se. If the patient's serum sodium is <130 mEq/L, Ringer's lactate solution (sodium concentration = 130mEq/L) may be used. It has the advantage of treating concurrent hypokalemia if present – correcting this often improves serum sodium. Otherwise, use Normal Saline (sodium concentration = 154 mmol/L) for volume correction.

### Sodium concentration in different solutions

- 0.9% NaCl: 154 mmol/L
- Ringer's Lactate: 130 mmol/L
- 0.45% NaCl: 77 mmol/L
- 5% Dextrose in water (D5W): 0 mmol/L

In general, patients with euvolemic and hypervolemic hyponatremia should be fluid restricted. For patients with SIADH and congestive heart failure, consider adding loop diuretics. For those with glucocorticoid deficiency, hydrocortisone should be administered. In these three conditions, a vasopressin receptor

antagonist may be considered in consultation with the inpatient specialists if the hyponatremia is refractory.

The amount and speed at which sodium is corrected must be determined before initiating replacement therapy. This can be calculated manually or via several online calculators, such as [Medcalc](#).

The rules of 6s and 100s can be used to avoid over-correction.

#### • Rule of 6s:

- “Six in six hours for severe symptoms, then stop.” If symptoms are not severe, then “six a day makes sense for safety.” In other words, for patients with severe symptoms, raise the sodium level by a maximum of 6 mEq/L over 6 hours. For those without severe symptoms, the maximum is 6 mEq/L over 24 hours.

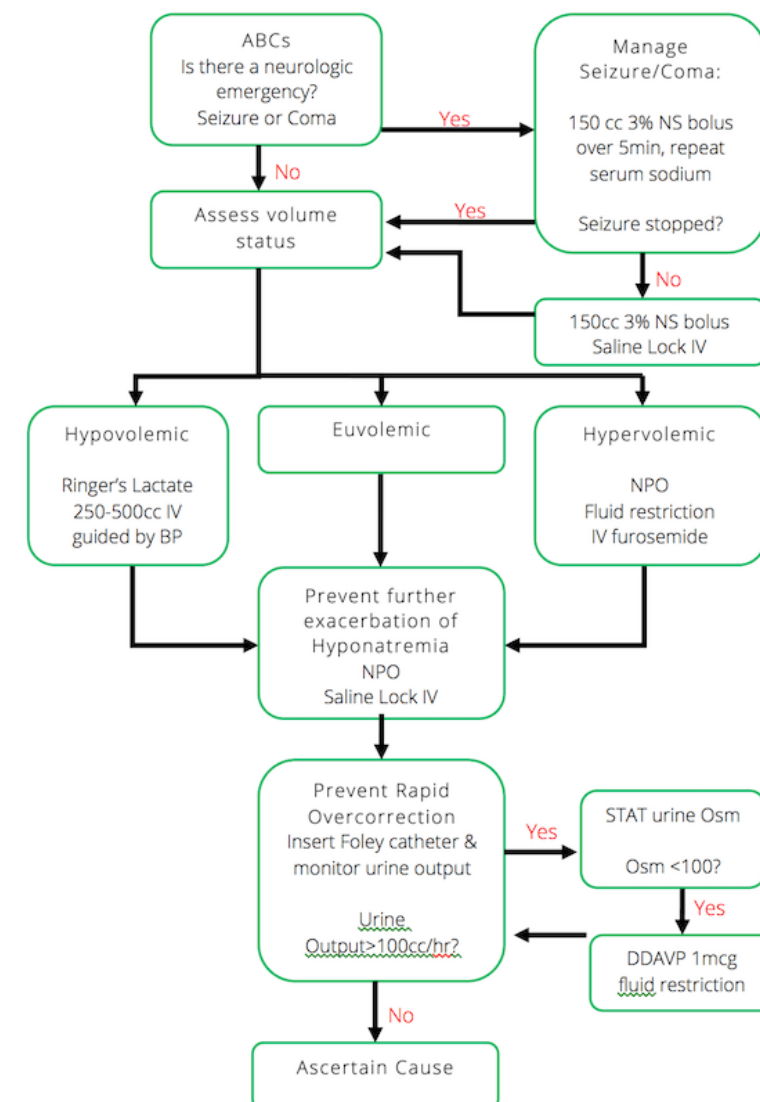
#### • Rule of 100s:

- If the output is more than 100 mL/hr, and the osmolality is less than 100 mosm/kg H<sub>2</sub>O, consider administering IV DDAVP 1 microgram to slow down the correction rate.

Rapid overcorrection is a risk factor for osmotic demyelination syndrome. As it has a predilection for the pons, it is also previously known as central pontine myelinolysis. Diagnosis is initially clinical, but later changes can be seen on the MRI. Clinical

presentation can take place after 2-6 days and may be irreversible. Symptoms range from ataxia and paresis to the “locked-in syndrome.” As the prognosis is poor, care should be taken to avoid this complication in the first place when correcting hyponatremia.

**Diagram 5.1**



*Treatment algorithm for hyponatremia (Conceived by Dr. Edward Etchells). Please read the whole article for more information ([link](#)).*

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

The most common cause of hyponatremia in the pediatric population is the gastrointestinal fluid loss (emesis or diarrhea) and inappropriate rehydration with a hypotonic solution. Ingestion of overly diluted formula and excessive water are other causes. Diagnosis and treatment are as described above.

Exercise-induced hyponatremia, a form of dilutional hyponatremia, may happen when patients consume excessive water or hypotonic liquids during exercise. Endurance sports and training, such as ultra-marathons, Ironman triathlons, and intensive military training deserve particular attention. Ironically, the patient may wrongly attribute the symptoms of lethargy (caused by hyponatremia) to dehydration, prompting further fluid intake. The management of these patients should follow the schema given above.

If hyponatremia is an incidental finding in an otherwise asymptomatic patient, water restriction can be advised until voiding occurs, to avoid development of symptomatic hyponatremia<sup>5</sup>

## Disposition Decisions

Patients with neurologic symptoms should be admitted to the ward or the ICU. Patients requiring excessive sodium correction should be placed under close monitoring.

Asymptomatic patients with mild hyponatremia from benign causes can be discharged with advice. They should be followed up by their primary physicians or referred to a specialist.

**References and Further Reading**, click [here](#)

# Hypoglycemia

---

by Rok Petrovčič

## Case Presentation

*A 75-year-old woman was brought to the emergency department (ED) by her relatives for “not being her usual self” for a day. She was on insulin therapy for her diabetes, but otherwise healthy.*

*On examination, she appeared confused and disoriented. Her vitals were as follow HR 95/min, RR 18/min, BP 141/85mmHg, T 37.7°C and SpO2 99% on room air. Given her past medical history, capillary blood glucose test was performed by the bedside. It was 2.6 mmol/L (47 mg/dl), and hypoglycemia was diagnosed.*

*She was given a bolus dose of intravenous glucose and much to the relatives’ relief and amazement; she returned to her normal behavior within 5 minutes. The patient herself reported lower urinary tract symptoms with a low-grade fever for the*



Audio is available [here](#)

*last two days. In addition, blood investigation showed that her renal function had also deteriorated significantly since her last primary care visit while continuing on the same insulin regime. The patient was subsequently admitted to a general ward for further evaluation and management.*

## Critical Bedside Actions and General Approach

Every patient who is critically ill or has any neurological derangement should have their blood glucose checked at the bedside. Blood glucose level is akin to a “vital sign” for any patient with neurological symptoms or signs, such as weakness, confusion, seizures or even coma. If the level of glucose is low (i.e., below 3,5 mmol/L), the patient should be promptly given glucose therapy, either orally or intravenously. The neurological deficit should reverse rapidly if hypoglycemia is the only reason for the deficit. The cause of the hypoglycemia should be investigated, and the patient monitored for recurrence of further episodes.

## Differential Diagnoses

Hypoglycemia can be iatrogenic or secondary to an underlying disease process. These include:

- Addison Disease
- Adrenal Crisis

• Cardiogenic Shock

- Hypopituitarism (Panhypopituitarism)
- Inadequate intake of food
- Insulin Resistance
- Insulinoma
- Poisoning
- Pseudohypoglycemia
- Renal failure
- Sepsis
- Stress

The above causes can apply to diabetic patients as well. In addition, one has to exclude inappropriate administration of diabetic medication, especially insulin. However, physicians should keep in mind that suicidal attempt with oral anti-diabetic agents can be more dangerous.

## History and Physical Examination Hints



Hypoglycemia must be excluded in any patient with coma, altered behavior, and any other neurological symptoms or signs. Signs and symptoms of hypoglycemia are combinations of neuroglycopenic and adrenergic effects:

- Neuroglycopenic symptoms are due to low glucose levels in CNS: blurred vision, weakness, tremor, seizures, paraesthesias, focal neurologic signs, and confusion.
- Adrenergic symptoms are tachypnea, tachycardia, sweating, hunger, headache, and anxiety.

Ask the patient about the time of last meal, exercise, and alcohol consumption. Detailed history on the use and dosage regime of diabetic medication is needed if the patient has diabetes. For patients with hypoglycemia secondary to a drug overdose, such as with oral hypoglycemic agents, suicide risk assessment has to be performed.

## Emergency Diagnostic Tests and Interpretation

Venous or capillary blood is checked with a glucose oxidase strip. If the level of glucose is  $<3.0$  mmol/L, take a venous blood sample for formal blood glucose level. The differential diagnosis and clinical picture direct additional diagnostic tests. When an overdose of diabetic medications are considered, additional studies such as serum insulin, C-peptide, cortisol, and glucagon may be indicated.

If neurologic/behavioral symptoms persist after treatment with glucose, evaluate for concurrent causes of altered mental status (mnemonic: “TIPS AEIOU”). A CT brain may be warranted.

A – Alcohol

E – Endocrine/Electrolyte/Epilepsy

I – Insulin

O – Overdose/opioids/oxygen

U – Uremia

T – Toxicologic/Trauma

I – Infection

P – Psychiatric/poisoning

S – Stroke / shock

## Emergency Treatment Options

Patients with hypoglycemia should be placed in a monitored area. The means of reversing the hypoglycemia depends on the patient’s mental status, ability to cooperate with oral intake, availability of intravenous access and medical and medication history.

- If the patient is conscious and can cooperate with oral intake, administration of food or liquid rich in simple carbohydrate (e.g., a sugary drink, sugar, candies, glucose tablets) is preferred.
- If the patient is unconscious or unable to cooperate with oral intake and intravenous access is available, give 50mL of IV dextrose 50% (equivalent to

25g of dextrose) over a few minutes. A second dose can be administered if the patient's mental status does not improve.

- If intravenous access is not available, IM/SC glucagon 1mg can be given. Glucagon takes a longer time to normalize mental status (around 7-10mins), and its effect tends to be short-lived. As glucagon raises blood glucose by releasing the hepatic glycogen reserve, it is not helpful in patients with depleted glycogen stores (e.g., liver failure or chronic alcoholism)
- For patients with sulfonylurea overdose, commence therapy with IV dextrose until the patient can tolerate orally. If episodes of hypoglycemia recur despite glucose therapy, consider the addition of SC octreotide 50-100 micrograms. Note that octreotide should only be used for recurrent sulfonylurea-induced hypoglycaemic episodes despite glucose therapy.

## **Pediatric, Geriatric, Pregnant Patient, and Other Considerations**

Children should receive 5 mL/kg of 10% glucose or 2.5mL/kg of 25% dextrose. Avoid using 50% dextrose in this population as it may easily result in thrombophlebitis.

Up to half diabetic pregnant patients on insulin will experience an episode of severe hypoglycemia during pregnancy. Careful titration of insulin is paramount to prevent recurrence of hypoglycemia while attempting to achieve optimal sugar control.

## **Disposition Decisions**

### **Admission criteria**

Patients with hypoglycemia generally require admission to an observation unit or the general ward, for evaluation and treatment of underlying cause and titration of diabetic medication.

Patients with unexplained or recurrent hypoglycemia should be admitted to a

monitored area. Consider consultation with toxicologist and psychiatrist for patients who overdose on their diabetic medication.

### **Discharge criteria**

The patient should only be discharged if the cause of the hypoglycemia is identified and deemed benign, have fully recovered, taking well orally and have no recurrence of hypoglycemic episodes after a period of observation. Discharge advice should be given.

### **Referral**

If discharged from the ED, the patients should be referred to their primary physician or specialist to follow up.

**References and Further Reading**, [click here](#)

# Thyroid Storm

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by Shabana Walia

## Case Presentation

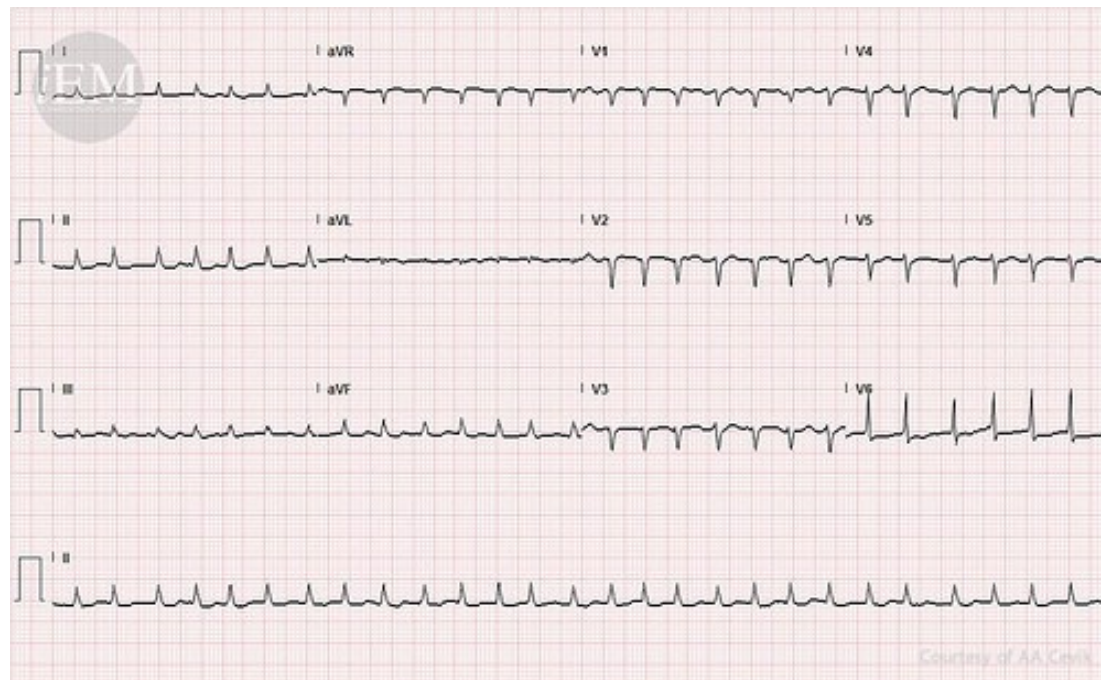
*A 68-year-old female with hypertension presented to the emergency department (ED) with worsening of lower extremity swelling for the last few months. She appeared to be confused over the last three days according to her husband. He also noted that she had a fever. She had intermittent chest discomfort and was feeling “anxious.” She was compliant with the prescribed antihypertensive (lisinopril and hydrochlorothiazide). She used no tobacco or illicit drug. She had a family history of hypertension and hyperthyroidism.*

*Her vitals at triage were as follows: BP 170/86mmHg, HR 136/min, RR 18/min, Temp 40.2°C and SpO2 100% on room air. She appeared agitated and flushed, with bilateral exophthalmos and lid lag. Her thyroid was diffusely enlarged with bruit noted. Her pulse was irregularly irregular. She had pitting edema up to the mid-shin. Bilateral plantar reflexes*

were 3+. The rest of the physical examination was unremarkable.

Bedside ECG is below

Image 5.2



Her blood test results were as follow:

Normal CBC and renal function.

Calcium: 11.5 mg/dL (8.6-10.2)

Thyroid stimulating hormone (TSH) < 0.01 mIU/L (0.34-5.6)

Free T3: > 30 pg/ml (2.5- 3.9)

Free T4: > 6 ng/dL (0.58-1.64)

Troponin: 0.10 (<0.04)

Pro-BNP: 3,000 pg/mL (0-100)

A diagnosis of hyperthyroidism was made, and she was evaluated for possible thyroid storm.



## Introduction

Thyrotoxicosis occurs when there is an excess of circulating thyroid hormone in the body, whereas hyperthyroidism refers to thyrotoxicosis that arises from a hyperfunctioning thyroid gland. Thyroid storm, a true endocrine emergency, is the most extreme form of thyrotoxicosis. It consists of a triad of severe hyperthermia, cardiovascular dysfunction and altered mental state. Although it occurs in less than 2% of patients with thyrotoxicosis, Emergency Physicians must maintain a high index of suspicion for thyroid storm because mortality approaches 80-100% if untreated. Prompt identification and appropriate treatment can reduce the mortality to 15-50%.

## Critical Bedside Actions

A patient with suspected thyroid storm should be placed in the resuscitation area. Evaluate the patient's ABCs and establish intravenous access. Vitals signs, including temperature, must be closely monitored.

Initiate aggressive supportive care, including temperature control. Treatment of the thyrotoxic state is aimed at inhibition of thyroid hormone release, inhibition of new hormone synthesis, inhibition of peripheral conversion of T4 to T3, and lastly blockage of peripheral beta-adrenergic receptors.

Evaluate the patient for precipitants (e.g., sepsis, noncompliance of anti-thyroid medications, trauma to the thyroid, radioactive iodine therapy, chemotherapy, recent surgery, and molar pregnancy) and complications of the thyroid storm (e.g., high output cardiac failure, atrial fibrillation). The underlying precipitant has to be addressed early and concurrently with treatment for complications of the thyroid storm.

## Differential Diagnosis

Differentials of thyroid storm include:

- Acute psychosis
- Alcohol or benzodiazepine withdrawal
- Anticholinergic overdose

• Encephalitis/meningitis

- Heat stroke
- Hypertensive encephalopathy
- Malignant hyperthermia
- Neuroleptic malignant syndrome (NMS)
- Sepsis
- Serotonin syndrome
- Sympathomimetic overdose

## History and Physical Exam Hints

As thyroid hormones act on almost every cell in the human body, thyroid storm will result in multi-organ dysfunction.

A thorough history and physical exam are keys to diagnosing thyroid storm. Patients often have a personal or family history of thyrotoxicosis. Initial symptoms may be vague and nonspecific. Symptoms of weight loss, ravenous appetite, emotional lability or irritation, and heat intolerance suggest

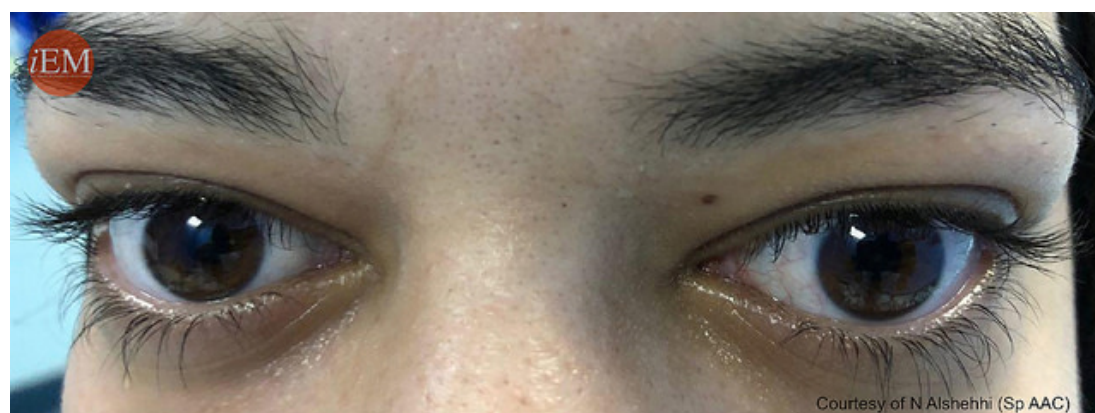


thyrotoxicosis. In a patient with prequel symptoms, hypermetabolic state, and deranged vital signs, the diagnosis of thyroid storm should be considered.

While the differentiation between thyrotoxicosis and thyroid storm is a clinical one, the diagnostic criteria by Burch and Wartofsky can help to identify a thyroid storm (modified in Table 5.2). A key diagnostic criterion is severe hyperthermia, as heat production becomes excessive and unregulated. Temperature above 38.5°C is common, and can even exceed 41°C. Patients also present with altered mental state and severe cardiovascular dysfunction.

Other common physical exam findings of thyroid storm include sweating, tachycardia, hypertension with a widened pulse pressure, anxiety, atrial fibrillation, tremors, exophthalmos (Image 5.3), goiter, and hyperreflexia. Note that the tachycardia seen in a thyroid storm is often out of proportion to the fever.

**Image 5.3**



**Table 5.2** Criterion for Diagnosing Thyroid Storm

CATEGORY	SITUATION	SCORE
Thermoregulatory Dysfunction	99-99.9	5
	100-100.9	10
	101-101.9	15
	102-102.9	20
	103-103.9	25
	104	30
CNS Effects	Absent	0
	Mild-Agitation	10
	Moderate-Psychosis, Delirium, Fatigue	20
	Severe-Seizures/Comatose	30
Gastrointestinal or Liver Dysfunction	Absent	0
	Moderate- Diarrhea/N/V/ Abdominal pain	10
	Severe-Jaundice	20
Cardiovascular Dysfunction	Tachycardia	5
	99-109	10
	110-119	15
	120-129	20
	130-139	25
	140	30
	Congestive Heart Failure	0
	Absent	5
	Mild-Pedal edema	10
	Moderate-Bibasilar rales	15
	Severe-Pulmonary edema	30
	Atrial Fibrillation	0
	Absent	10
	Present	30
	Precipitant History	0
	Negative	10
	Positive	30

Criterion for diagnosing thyroid storm, Modified from the original Burch HB, Wartofsky L. Criteria: Life-threatening thyrotoxicosis: Thyroid storm. Endocrinol Metab Clin North Am 22:263-277, 1993. Please read the article for more information.

**SCORING SYSTEM** adds total points based on patient's history and physical exam:

- >45: Highly suggestive of thyroid storm
- 25-44: Suggestive of an impending thyroid storm or thyrotoxicosis
- <25: Unlikely thyroid storm based on presentation

**The patient in our case presentation above**

- Temp: 30
- CNS effects: 10
- GI-Liver: 0
- Tachycardia: 20
- CHF: 5
- Atrial Fibrillation: 10
- Precipitant History: 0
- TOTAL POINTS: 75

## Emergency Diagnostic Tests and Interpretation

The following investigations are indicated in patients with suspected thyroid storm: CBC, renal function test, liver function test, thyroid function tests, metabolic panel (including calcium), ECG, CXR.

While confirming the presence of thyrotoxicosis, thyroid function tests alone cannot be used to rule in or rule out thyroid storm reliably. Thyroid stimulating hormone (TSH) will be low or undetectable, with elevated free triiodothyronine (T3) and its prohormone, thyroxine (T4). Hypercalcemia, elevated alkaline phosphatase, and hyperglycemia are other common lab abnormalities seen due to bone resorption, bone remodeling, and glycogenolysis, respectively. Abnormalities on ECG include premature atrial, premature ventricular contractions, atrial fibrillation, or atrial flutter. The CXR may show cardiomegaly and pulmonary vascular congestion – indicating heart failure. If global or focal neurological deficits are found, it is reasonable to

consider a head CT scan to rule out a precipitating or concurrent intracranial process.

Use the Burch and Wartofsky criteria to evaluate for the likelihood of thyroid storm. If the diagnosis is suspected, treatment with the medications listed in Table 2 should be initiated. The primary goals are blocking the peripheral effects of thyroid hormone, preventing the synthesis of T3 and T4, and inhibiting the release of preformed thyroid hormone.

## Emergency Treatment Options

Initiate supportive care expeditiously. Administer intravenous fluid to correct volume depletion, supplemental oxygen for hypoxia and external cooling measures for severe hyperthermia. Acetaminophen alone is not helpful as the hyperthermia in thyroid storm is not a central, hypothalamic regulatory problem. Cooling blankets, ice packs or even cold intravenous fluids can be used. In patients with airway compromise, rapid

sequence intubation and paralysis should be considered with a secondary aim of temperature control. Aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided as they can increase peripheral free T3 and T4 due to their protein binding properties.

**Table 5.3** Medical Treatment for Thyroid Storm

GOAL	MEDICATION	MECHANISM OF ACTION	EXTRA CONSIDERATIONS
STEP 1: Block peripheral adrenergic effects of thyroid hormone	<p>Propranolol: 60-80mg PO q4hrs or 0.5-1 mg IV q1h (slow infusion)</p> <p>Esmolol: 250-500micrograms /kg IV bolus, then 50-100micrograms/ kg infusion</p>	<p>Beta blockade, shorter half life with esmolol</p> <p>Propranolol has the additive mechanism of blocking conversion of T4 to T3 in peripheral tissues</p>	<p>Use propranolol with caution in those with signs and symptoms of congestive heart failure, as this can cause cardiogenic shock and collapse</p> <p>Esmolol is a selective B-1 blocker, thus can be used in patients with bronchospasm or asthma</p>
STEP 2: Prevent synthesis of thyroid hormone	<p>Propylthiouracil (PTU): 600-1000mg PO initial dose, then 200-250 q4h</p> <p>Methimazole: 20-30mg PO initial dose, then 20-30 q6h</p> <p>Dexamethasone: 2mg IV q6h</p> <p>Hydrocortisone: 300mg IV initial dose, then 200 mg q8h</p>	<p>Central mechanism of PTU and MMI: Inhibits thyroid peroxidase. Thyroid peroxidase oxidizes iodide to create iodine, which is then added to tyrosine residues on thyroglobulin, creating T3 and T4</p> <p>Steroids decrease the conversion of T4 to T3 in the periphery</p>	<p>PTU also inhibits peripheral conversion of T4 to T3 by inhibiting the enzyme 5'deiodinase</p>
STEP 3: Inhibit release of thyroid hormone	<p>Lugol's Solution (Iodine): 8 drops PO q6h</p> <p>SSKI: 5 drops PO q6h</p>	<p>Decreases the release of thyroid hormone from the thyroid gland</p>	<p>Must be given at least one hour after PTU or it can increase thyroid stores by iodinating tyrosine</p>

Table produced by author.

## Pediatric, Geriatric, Pregnant Patients And Other Considerations

Pediatric patients may not present with the classic symptoms of hyperthyroidism. They may only demonstrate jitteriness, agitation, restlessness without typical ophthalmologic or other systemic findings.

The presentation in geriatric patients may be even more subtle. These vague complaints may be easily dismissed by the patients themselves or care provider, or even mistaken as normal aging. Alternatively, they may also present with primarily cardiovascular complaints, such as palpitations, chest discomfort or signs of heart failure. Therefore, a high index of suspicion should always be maintained in the elderly.

Pregnancy can trigger thyrotoxicosis in up to 10% of patients, even up to 6 months postpartum. High estrogen and human chorionic gonadotropin level have weak TSH-like effects and contribute to an overall hyperthyroid state. Propylthiouracil (PTU) is preferred over methimazole (MMI) in this population, as it does not cross the placenta as readily. Methimazole is also associated with choanal atresia and aplasia cutis. Both drugs are FDA category D in pregnancy.

## Disposition

Admit all patients with thyroid storm to the ICU or high dependency unit for ongoing monitoring and treatment.

Admit the patients with partially-controlled thyrotoxicosis symptoms for further management. Patients with mild controlled symptoms and stable vital signs may be discharged with close follow up with the primary care physician or endocrinologist.

**References and Further Reading**, click [here](#)



## Chapter 6

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# Selected Environmental Emergencies





# Burns

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by Rahul Goswami

## Introduction

The skin is the largest organ in the body. Its physiological purpose is to protect the body contents from foreign pathogens and maintain thermoregulation.

The skin layers consist of:

- Epidermis – outermost layer containing epithelial cells
- Dermis – middle thicker layer comprising connective tissue, nerve endings, blood vessels and sweat glands.
- Subcutaneous tissue – lies just above the muscle layer and contains fat cells and connective tissue.

Damage to the skin can be temporary or permanent, and a common

mechanism is burns. This chapter will look at the different types of mechanisms along with their assessment and management.

## Thermal Burns

When excessive heat is applied to the skin, the destruction of the tissue is known as a thermal burn. This may involve inflammation and healing but if enough heat is applied, there is coagulative necrosis and that area of skin is dead. The type of burn depends on many factors and only careful assessment with appropriate treatment can lead to good outcomes.

**Image 6.1**



## Assessment

- Depth
  - How deep the injury is can be estimated from the surface condition as well as the mechanism.
- Area
  - The area affected is a good predictor of outcomes. Here is a [calculator](#) to remind you of the importance of surface area burnt for survival. Most calculators are only applied to 2nd and 3rd-degree burns.

- Estimation of the area can be done in the following ways:
  - **Rule of Nines.**
  - Palm area estimation – This utilizes the fact that the palm (including fingers) of patients is approximately 1% of the patient's body surface area. Estimate how many “palms” is burnt to get the approximate total burn surface area % (or TBSA). This is especially useful in children.
  - Pediatric assessment is more difficult as their surface area is unlike adults. This [link](#) is helpful.
- Location
  - certain areas of the body are more susceptible to edema and constriction and hence aggressive management is warranted. Examples include:
    - Genital burns – fertility consequences
    - Facial burns – cosmetic and psychological sequelae
    - Ocular burns – lasting visual impairment
    - Airway burns or inhalational burns – this requires eventual airway protection as the swelling will occlude the ability to breathe in a few hours
    - Special consideration in the assessment of burns in children must be given to Non-Accidental Injury (NAI) or child abuse. Certain patterns of burns are suspicious in nature and if seen, should alert the physician to the safety of the child. Examples of these patterns include:

- Glove or stocking distribution indicating forced immersion
- Cigarette marks or other implement contact marks

## Management

1. First aid – the primary treatment is cooling the burn area with running water. This not only gives pain relief but also halts the thermal coagulative process and prevents more area of skin being damaged. Following this, burns should generally be wrapped in dry cling film or dressing before transport to a medical facility. This reduces pain from surrounding clothes and wind. The author was once asked to explain minor injuries including burns and this is the [video](#) for it.
2. Analgesia – simple oral analgesia (paracetamol or NSAID) followed by opioids if need be. This is the most important step in hospital. This is a good [video](#) for the different types of

**Table 6.1** Burn Degrees

DEGREE	EXAMINATION FINDINGS OF SKIN	EXAMPLE
1st degree or superficial	Red, painful	Sunburn
2nd degree is divided into: - superficial partial thickness - deep partial thickness	Sometimes blistered, painful, moist	Scald or flash burn
3rd degree or full thickness	Dry, white, insensate, leathery and decreased sensation	Flame or immersion
4th degree or deep full thickness	Damage extends to fat, muscle or bone	Chemical or electrical

treatments, applications, and dressings available.

3. Fluids – When skin is lost, a large amount of fluids may leak into the injured space and also evaporate. Regimens may vary but a good rule to follow is the Parkland formula. Here is an [online calculator](#) for this. In essence, give in mls:

- $4 \times \text{weight of patient} \times \text{TBSA burnt (\%)}$

- Give 1/2 of the total in 1st 8 hours, and then give 2nd half over next 16 hours
- Hartmann's solution is the ideal replacement fluid
- Pediatric patients require even more specific control of fluid replacement and have their own formulae.

4. Specific dressings and burns ointments have changed over the last decade. Here's a good, updated [site](#) for such information.
5. Blister management – whether to remove or puncture the blister or leave it alone has been controversial for a long time. The [current consensus](#) is to de-roof them.
6. ATT – Anti-tetanus toxoid for anyone not immunized or whose last dose was more than 10 years ago.

## Referral to the Burn Unit

The burn unit is a specialized unit which deals specifically with burns patients' needs (dressing changes, escharotomy, debridement, physio, etc.). They are essential units which provide specialist care and dedicated rehabilitation.

Criteria for referral vary between countries but in general patients who require referral or review include:

- Total Burn Surface Area (TBSA) greater than 10% in adults
- TBSA greater than 5% in children
- Full thickness burns of any area
- Circumferential burns of limbs or chest/abdomen
- Facial burns or inhalational burns
- Scrotal/genital burns

- Airway burns (if the unit has an ICU built in)

Examples of units are in these links ([1](#), [2](#))

## Electrical Burns

Electricity contains a large amount of energy, and when passing through the body, it causes damage along its path and usually manifests itself as a burn at the entry and exit points.

**Image 6.2** Image shows the entry wound of an electrical injury





**Image 6.3** Image shows the exit wound on the leg.



There are a few modes of injury:

- Power points – The amount of damage caused by home/industrial mains depends on contact time, voltage and current.
- Lightning strikes, on the other hand, are rare but cause devastating injuries.
- A new phenomenon these days might be TASER burns in patients shot by police.

## Assessment

A similar approach to assessment as thermal burns but beware of the lack of area affected. Electrical energy arcs into

the body at small points but does tremendous damage along the route of the current all the way to the exit point. Thus a careful scan of the body and an ECG must be done to elicit damage. Muscle damage leads to breakdown called rhabdomyolysis and this, in turn, leads to renal failure and multi-organ failure if not treated promptly. Shoulder dislocations from being jolted from the electricity and head injuries are also common.

Lightning strikes have far more energy transmitted than household mains. Lichtenberg flowers are classically (but rarely) seen in lightning strike burns. Here lies a good write-up and picture of it.

**Image 6.4** Image shows urine of the patient who has rhabdomyolysis after electrical burn.



## Management

ED management includes:

1. Analgesia – simple oral tablets to IV opioids depending on pain score
2. Fluids – to prevent and treat renal failure



3. Bicarbonate – in cases of rhabdomyolysis, alkalization of the urine will help draw products of muscle break down out of the body.
4. Treat other injured joints or organs sustained from the jolt when shocked (e.g., shoulder dislocations)

**Image 6.5** Escharotomy in a severely injured patient with high voltage electricity



## Chemical / Radiation Burns

**Image 6.6** Image shows chemical burn on the hand.



**Image 6.7** Image shows chemical burn on the foot.



Like most toxic ingestions and exposures, the extent of injury depends on:

- Type of toxin
- Concentration of toxin
- Length of exposure to it
- Immediate decontamination and first aid

A good explanation of chemical burns from common household items can be found [here](#).

Although **radiation burns** sound very ominous, they are handled similarly as all other burns. The myth that exposed patients can contaminate the whole hospital is unjustified and applies to only a specific scenario involving “dirty bombs.”

Most common radiation burns are actually from medical facilities such as cancer treatment or x-ray imaging centers as well as tanning booths and

lamps. A good explanation of radiation damage and risks can be found in this [pdf](#).

## Assessment

A similar calculation for TBSA burned can be used as in thermal burns shown in the section above. However, the location of the burn and concentration of the agent have far more impact.

For example:

- Alkali burns of the cornea can result in permanent blindness
- Hydrofluoric acid burns can result in systemic fluorosis which is life-threatening
- Corrosive burns to the esophagus can cause permanent swallowing difficulties

Hence a thorough examination is more important than any lab or imaging test. Geiger counters can be used to detect contaminated patients exposed to radionuclides. This needs to be done by experts in the field.

## Management

Probably the most important interventions to get right are the first aid processes:

- Removal of offending agent
- Irrigation with water to dilute and neutralize
- Transport to a medical facility

ED management includes;

1. Analgesia – IV opioids most commonly as they are deep dermal burns
2. More irrigation of the affected area if symptoms or pain persists
3. Irrigation of the eyes is of utmost importance and the only way of removing the offending agent.
4. Local anesthetic also helps during irrigation. A good video of this is [here](#).
5. Anti-tetanus toxoid
6. Specific antidotes:



- Hydrofluoric acid – this acid is one of the most corrosive known and its systemic effects lower calcium to a life-threatening level. Hence calcium is essential not only for cardiac stability but also for analgesia. Calcium can be given in oral, topical gel and IV forms.
- Radionuclide poisoning – decontamination is once again dealt very comprehensively in this pdf.

6. Referral to the burn unit

## Inhalation injuries

Image 6.8



### Thermal inhalational injury

- Airways can become swollen due to inflammation. Prompt airway protection measures need to be initiated before the swelling becomes too severe (impairs breathing)
- Tracheobronchial edema / inflammation

• Acute Respiratory Distress Syndrome (ARDS) or acute lung injury

### Smoke inhalation

- Particulate matter in smoke causes airway and bronchial inflammation which can lead to pulmonary edema, bronchospasm, and even ARDS. Signs of such injury include soot in the oropharynx, singed nasal hairs, hoarseness, stridor or confusion/agitation. Watch this [video](#).

### Chemical inhalational injury

When patients are thermally injured, the environment they were in can cause harm as well. Here are two of the most common inhalation injuries that patients trapped in burning environments receive.

- CO poisoning – This gas is a by-product of combustion. Symptoms can range from confusion to coma. The only way to detect it is a high degree of suspicion and via arterial blood gas. The treatment is 100% oxygen. In some cases with neurological symptoms and

coma, hyperbaric oxygen therapy is recommended.

- CN poisoning – This gas is produced when furniture and other plastics are combusted (usually house fires). It is a pulmonary irritant and can cause tissue hypoxia which may lead to cardiac arrest. Treatment is decontamination, oxygen and specific antidotes (not in the scope of this section).

**References and Further Reading**, click [here](#)

# Drowning

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by Ana Spehonja

## Case Presentation

*A 6-year-old previously healthy male was brought to the emergency department (ED) after he fell into a freshwater lake while playing on the dock. Eyewitnesses found his body floating face down in the water. He was unaccounted for 10-15 minutes. They started basic life support right after they pulled him out of the water. He was cyanotic, apnoeic, pulseless with fixated and dilated pupils and tympanic temperature of 26,7 at arrival to ED. CPR was continued. After established airway and assessment for other injuries, they began to warm him up. ABG showed combined respiratory and metabolic acidosis with severe uncorrected hypoxemia. 15 minutes post mechanical ventilation and the return of a spontaneous heart rate with adequate blood pressure 110/67, SpO2 was 96%, pupils small and reactive to light and the tympanic temperature was 32,2°C. There was no spontaneous*



*respiratory effort. ABG analysis showed uncompensated metabolic acidosis with corrected hypoxemia. He was stable enough for transfer to Paediatric ICU. After two days, ICU reported him to be stable with normal temperature.*

## Definition and Terminology

### Previous Definitions

Drowning – submersion injuries that resulted in death in under 24 hours

Near drowning – those that survived over 24 hours.

Dry drowning – a state caused by laryngospasm, followed by convulsion, hypoxia that leads to loss of consciousness and death without entry of fluid to lungs. It accounts for 10-20% of submersion injuries.

Wet drowning – is a state caused by the aspiration of water. This leads to dilution and washout of the pulmonary surfactant. The result of this situation is diminished gas transfer across the alveoli, atelectasis, and ventilation-perfusion mismatch.

Currently, all submersion injuries are defined as drowning. In 2005 World Health Organisation published a new policy defining

drowning as the “the process of experiencing respiratory impairment from submersion/immersion in liquid. Drowning outcomes are defined as death, morbidity, and no morbidity.”

*Drowning happens due to closed glottis, hypoxia, and cardiac arrest.*

*People don't inhale water; it gets into the lungs later.*

**Immersion syndrome** is syncope resulting from cardiac dysrhythmias on sudden contact with water that is at least 5°C lower than body temperature. Vagal stimulation leads to asystole and ventricular fibrillation secondary to QT prolongation after a massive release of catecholamines on contact with cold water. Loss of consciousness leads to secondary drowning. We can prevent this from happening with wetting our face and head before entrance into the water.

Drowning incidence is most common in toddlers and young children as they can drown in toilets, buckets, and bathtubs. The second are adolescents and young adults and third are the elderly.

The fluid medium in which a submersion happened has little clinical relevance, pulmonary injury and hypoxia are caused by the amount of water aspirated and the duration of submersion. In

both cases, the effect of the osmotic gradient on the very delicate alveolar-capillary membrane increases its permeability and exacerbates fluid, electrolyte shifts and plasma.

Rapid CNS cooling before significant cardiac dysrhythmia provides cerebral protection in cold water submersion.

In submersion victims amount of swallowed water is much greater than aspirated, as a consequence 60% of patients vomit after a submersion event. Well-known complication with aspiration of gastric contents is pulmonary injury and increased possibility for acute respiratory distress syndrome.

### *Type of the water does not matter*

When the patient is rescued alive is the clinical picture determined predominantly by the amount of water has been aspirated and its effects. Osmotic gradient effects the very delicate alveolar capillary membrane increases its permeability and exacerbates fluid, plasma and electrolyte shifts.

We should also consider the precipitants of submersion injury which may be drugs or ethanol intoxication, cardiac arrest, hypoglycemia, seizure and attempted suicide or homicide.

## **Prehospital Care**

The most important thing is early CPR, as it optimizes the outcome. This is the reason why there is a need to train laypersons in CPR.

If the patient is notbreathing give 5 rescue breaths immediately, followed by 30 chest compressions and continuing with 2 rescue breaths and 30 compressions until signs of life reappear. Positive pressure bag-valve-mask ventilation should be administered.

If the heart is beating give only breaths, not CPR. Victims with only respiratory arrest usually respond after a few rescue breaths.

If the patient is spontaneously breathing, let him cough, place him in the recovery position and administer high-flow oxygen mask (15 liters of oxygen per minute).

### *If it is possible, resuscitate in the water (ventilation alone)*

Endotracheal intubation and positive pressure ventilation are necessary if there is no recovery of spontaneous respiratory effort.

Obligatory transportation to ED is for the patients who have a loss or depressed consciousness, an observed period of apnoea and those who require a period of artificial ventilation.

## **Emergency Department Care**

First steps are assessing and securing the airway, providing oxygen, determine core temperature and assisted ventilation as necessary.

If the patient is in cardiac arrest you should follow ACLS guidelines.

The following management shows the steps when the patient is not in cardiac arrest:

### A. Airway

- When the pulmonary examination is abnormal or there is oxygen requirement, admission or transfer to monitored bed is needed.
- When the GCS is under 13 the patient should receive supplemental oxygen and ventilator support if needed.
- The patient should be intubated when high-flow oxygen cannot maintain an adequate partial pressure of arterial oxygen.

### B. Breathing

- Arterial saturation should be between 92-96%. Treatment should be administered like ARDS: add PEEP, low tidal volumes, and permissive hypercarbia.
- Supranormal levels of positive end-expiratory pressure are beneficial to recruit fluid-filled lung units. Special consideration should be taken to avoid lung over-distention and ventilate-associate lung trauma.

- Avoid suction as it disrupts oxygenation and do not extubate early (lung injury may present later). Medications should not be administered through the endotracheal tube.

### C. Circulation

- We are looking for the presence of significant dysrhythmias, QT prolongation or ischemia with cardiac monitoring and an electrocardiogram.
- If there is cardiopulmonary arrest or asystole when the patient comes to ED, we should consider discontinuing resuscitation efforts, because of profound neurologic handicaps.
- There was not shown benefit in efforts to control cerebral edema, with use of mannitol, loop diuretics, hypertonic saline, fluid restriction and mechanical hyperventilation.
- Continuous cardiac monitoring, pulse oximetry, temperature monitoring.

### Varieties of tests in the ED include

- Gastric tube, laboratory studies (frequent arterial blood gas measurements)
- Chest radiography (initial chest radiographs may be unremarkable, even in the setting of severe and evolving pathologic process)

## Risk Groups

- Drowning is the leading cause of injury mortality in children 1 to 4 years of age.
- Children can develop dilutional hyponatremia and seizures in freshwater near-drowning.
- Children have hypothermia more quickly because of a lower ratio of body mass to surface area. There was no shown benefit in controlled hypothermia, barbiturate coma, and intracranial pressure monitoring.

## Prognosis

There is no prognostic scale that accurately predicts long-term neurologic outcome. There is documented normal neurologic recovery even with fixed and dilated pupils, cardiovascular instability, prolonged submersions and persistent coma. Complete recovery within 48 hours is expected if there was no need for cardiopulmonary resuscitation on the scene or in ED. If there are no continuous neurologic and cardiovascular deficits shown, the patients should recover completely. Those who needed CPR in ED have a poor prognosis, because of significant anoxic or ischemic insult to the brain and other vital organs.

## Disposition Decisions

Apnoea, hypoxia, unconsciousness, dysrhythmia or abnormal chest radiograph are signs for admission.

If ED resuscitation/CPR was required the patient should be admitted to the intensive care unit for continuous cardiopulmonary and frequent neurologic monitoring.

The patient can be discharged after 4-6 hour observation period if the Glasgow Coma Scale is 15, oxygen saturation over 94% on room air and if pulmonary examination does not reveal rales, rhonchi, wheezing, or retractions. We should warn the patient to return if mental status changes, pulmonary symptoms or fever occur.

**References and Further Reading**, click [here](#)

# Heat Illness

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by Abdulaziz Al Mulaik

## Case Presentation

*A 57-year-old male is brought to the emergency department by EMS during Hajj. The patient as stated by the paramedics was “found face down” in the street under direct sunlight, where outside temperature is 45°C. Initial vitals are BP: 91/55, HR 130 . O2Sat 95% on room air, RR 25 , Axillary temperature 39°C, and his Glucose check was 8 mmol/l. On examination, the patient is not oriented nor alert but he moans to painful stimuli, and he is maintaining his airway with no drooling. You ask a member of your team to repeat temperature measurement rectally, and he finds it to be 42.3°C. The rest of your physical examination is unremarkable. You remove all of the patient’s clothes and spray him with lukewarm tap water, you then turn on a fan and raise the head of the bed and the side rails. A continuous temperature probe is inserted rectally. A cardiac monitor with pulse oximetry is connected, and*



*blood samples were drawn for laboratory testing. After reaching a rectal temperature of 39°C, you direct your team to dry him and cover him with a light bed sheet. On subsequent examination, the patient is conscious, alert and oriented. Vitals are HR 105, O2Sat 96% on room air, RR 20. Labs reveal multiple abnormalities including respiratory alkalosis and elevated liver enzymes. Your disposition includes appropriate medical consultation and admission to a medical ward for further management.*

Basics of Heat Transfer is explained in this [video](#).

## **Critical Bedside Actions and General Approach**

Heat stroke is a devastating disease that might have permanent sequelae if lifesaving interventions are delayed. It is defined simply as a failure of thermoregulatory mechanisms to cope with either internal heat production known as exertional heat stroke (EHS) or external environmental heat, known as classic heat stroke (CHS). Watch this [video](#).

To diagnose a heat a stroke, the patient has to have a Central Nervous System (CNS) impairment and core temperature of more than 40°C. The spectrum of neurological abnormalities ranges from mild confusion to full-blown coma with GCS of 3. Core temperature has to be measured and continuously monitored using rectal or esophageal probes as peripheral measures of temperature are unreliable and does not correlate with core temperature.

The essence of heat stroke management is to “Cool first, then ask questions!”, as it is a time-sensitive condition, where cooling takes precedence over everything else including confirmation of the diagnosis. ABCs are the way emergency medicine practitioners approach every patient rightfully, so, to ensure that all critical decisions are made in a timely fashion. Heat stroke is not an exception to this role, as the disturbance in consciousness could result in significant airway complications. A complete

airway assessment should be immediately performed when the patient arrives in the emergency department while cooling measures being set up. Other physical examination details are important but should not delay cooling.

Two important observations are worth taking note of: the first relates to how difficult intravenous peripheral access might be during early stages of resuscitating in a dehydrated victim which is going to be compounded by cooling as this will result in peripheral vasoconstriction. The second is a phenomenon related to excessive watery diarrhea during cooling, and this mandates adequate preparation by the nursing and janitorial team. There are two possible explanations for this phenomenon; the first is the compensatory vasoconstrictive changes to the splanchnic vasculature as blood pools peripherally for cooling. The second proposed mechanism is the multisystem failure caused by heatstroke which includes the gastrointestinal system where epithelial cells shed and propagate an inflammatory diarrhea.

## Differential Diagnoses

In a febrile illness, be it infectious or otherwise, circulating pyrogens resets the normal temperature in the thermoregulatory control center to a new set point above normal. The entire thermoregulatory pathways in the body will work to achieve the new set point; hence febrile patients will have behavioral changes where they will seek warmer environments. Cooling febrile patients by cold towels or showers has a mild effect as the body

will continue warming up to the new set point. No harmful effect is exerted directly by fever alone, and clinical focus should be directed toward the cause of the fever.

Heat stroke, on the other hand, is a failure of the thermoregulatory system to cool the body. Therefore, all antipyretics are potentially harmful interventions as they work primarily on thermal set point reduction which is already normal in heat stroke patients. Reverting patients to a normal thermal level will reboot the thermoregulatory system, hence the urgency of cooling.

Minor heat illnesses are expected in the right environmental conditions. These illnesses are considered the milder side of the spectrum of heat illnesses with heat stroke on the other side. A list of these disorders with their classic presentations and recommended management strategies can be found below (Table 1).

**Table 6.2** The list of minor heat illnesses with their clinical features and treatment

MINOR HEAT ILLNESS	MOST PROMINENT CLINICAL FEATURES	TREATMENT
<b>Prickly heat</b>	Very pruritic vesicular rash on an erythematous base	Chlorhexidine lotion
<b>Heat syncope</b>	Standing in heat for long time with no previous acclimatization to heat	No specific treatment required
<b>Heat cramps</b>	Muscular cramps AFTER working in heat	Oral 0.1% salt solution
<b>Heat edema</b>	Swollen feet and ankles in healthy patients after standing in heat for long time	No specific treatment required

## History and Physical Examination Hints

Situational awareness is a vital skill to emergency physicians, as one should be aware of high ambient temperatures and high humidity days as they are perfect conditions for classical heat strokes. Lonely elderly community members with low socioeconomic status are particularly vulnerable to CHS as they have poor access to good air conditioning and ventilation. Usage of some medications which impairs adrenergic response to heat

by sweating is another risk factor for developing heat illness. Generally speaking, CHS is not common in geographical areas where average temperature throughout the year is high, as communities living in these places will develop behavioral tactics to avoid the heat.

Intense exercise, military training, sports competitions or prolonged labor might induce another type of stroke known as exertional heat stroke (EHS), which differs from CHS in laboratory indices and long-term complications. A third less common type of heat stroke is confinement hyperpyrexia where the patient is exposed to moderately high ambient temperatures for a long time.

Measuring body core temperature is perhaps the most important physical assessment, whenever heat illnesses are considered in the differential diagnosis of a given patient. Peripheral temperature measurements correlate poorly with core temperature. The two methods of measuring core temperature are either through the esophagus or the rectum with the latter representing the majority of clinical practice. A common pitfall in using rectal temperature is inserting the probe to an insufficient depth which will render readings to be inaccurate in both directions especially if ice packs have been applied to the groin. Rectal probes, in general, have to be inserted 15 cm inside the rectum to mitigate the effects mentioned above, but manufacturers may recommend different depths.

Tachycardia and hypotension are commonly seen and represent the physiologic response to heat, as peripheral vascular resistance decreases to allow the blood to be cooled at the surface of the skin, leading to a high output status. This phenomenon might explain other heat illnesses like heat syncope and heat edema.

Heat stroke is a multisystem disease affecting almost every organ in the body. CNS effects might range from simple confusion to deep coma. Seizures, in general, are common and might be confused with shivering during cooling, but both disorders need to be treated, the former for neural protection and the latter to prevent heat generation. Heat stroke patients might have derangements in their hemostasis represented clinically as melena, hemoptysis, conjunctival hemorrhage or epistaxis. Prickly heat patients will have pruritic vesicles on an erythematous base. These vesicles are sweat glands with blocked pores by macerated stratum corneum.

## Emergency Diagnostic Tests and Interpretation

EHS and CHS have different derangements in laboratory studies with some similarities. For instance, respiratory alkalosis is a physiologic response to heat stress, which is profoundly represented in CHS, whereas lactic acidosis is the prominent acid-base disturbance in EHS. Moreover, liver enzymes should be elevated in both EHS and CHS with numbers in the tens of thousands above normal cutoffs, to the degree that their elevation is a cardinal diagnostic criterion, and their absence will render the diagnosis of heat stroke unlikely. Another common difference between CHS and EHS is glucose level as it might be low in the latter but not the former.

## Emergency Treatment Options

The fastest way to transfer heat and to cool patients is through conduction, which is the direct transfer of heat between molecules. Full body water

immersion can do this, and although this is theoretically the best cooling method, it is clinically challenging as it poses a risk of aspiration and renders patient's accessibility quite difficult. Convection, which is the thermal loss due to gas movement around the body, combined with evaporation can achieve the similar speed of cooling to full body immersion. This combination can be achieved by spraying the patient with lukewarm water followed by fanning with warm air. Mist fans are very convenient and have the added benefit of their ability to fan multiple patients at once. Cooling units with intermittent water sprays from all directions around the patient are costly and not available in most hospitals, and they have recently fallen out of favor, even in Hajj despite their availability, mainly due to safety concerns as they limit access to patients.

Invasive cooling procedures such as cool IV fluids have not been proven to change the outcomes as their evidence remains to be weak. On the other hand, thoracic,



bladder, rectal and peritoneal lavage should only be used when all other measures fail. We suggest abandoning them even in the very sickest of patients, as a neurologically meaningful recovery is highly unlikely.

**Image 6.9** monitor showing the current vitals while the patient is cooled.



A protected airway should be maintained in heatstroke patients, and efforts should be taken to resist the urge to intubate them. Intubating solely for the low level of consciousness is ill-advised and will expose patients to unnecessary adverse effects of intubation. With that being said, airway protection is of utmost importance

and should take priority on any other diagnostic or therapeutic procedures.

Peripheral blood pooling is in the heart of heat stroke pathology, so hypotension is common in these patients and fluid administration should be very judicious as the blood pressure usually will pick up as the core temperature drops down. Aliquots of 250 cc of crystalloids should be used when fluids are needed, and repeated dosing should take place after volume status assessments.

## Special Patient Groups and Situations

Pediatric heat stroke patients are usually victims of confinement hyperpyrexia. Attention should be paid if further interventions are needed to protect a child from abuse or maltreatment. Geriatric patients, on the other hand, are usually the victims of CHS. Emergency physicians should play their role in advocating for closer community ties and socioeconomic support by appropriate authorities for those patients.

A mass casualty incident of heat stroke and heat exhaustion patients should be expected in preparing for a mass gathering event and mitigation measures should be sought in advance. Public education to seek shade, drink enough fluids, use umbrellas and installing mist

**Image 6.10** The row of beds with mist fans in a sunstroke unit. A cooling unit can be seen at the far right.



**Image 6.11** Fiberglass grooved beds with waterproof mattresses in a sunstroke unit.





pipes, venting fans, cooling stops in a path of a riot or a race are few examples.

## Disposition Decisions

Heat stroke patients usually require higher care upon admission as their stability is not certain and further assessment of heat stroke complications should take place in the hospital. Stable, conscious heat exhaustion patients may be discharged with education and close follow up. Minor heat illnesses should be treated as a case by case scenario, but they rarely require inpatient care.

**References and Further Reading**, click [here](#)

# Hyperthermia

by Puneet Sharma

## Introduction

Hyperthermia is elevated body temperature. It generally due to failed thermoregulation which occurs when a body produces or absorbs more heat than it disseminates. Extreme temperature elevation then becomes a medical emergency requiring immediate treatment to prevent disability or death.

Temperature greater than 37.5–38.3 °C (99.5–100.9 °F) depending on the reference used is, hyperthermia, with severe hyperthermia being greater than 40°C(104°F). Different sources have different cut-offs.

Hyperthermia differs from fever in that the body's temperature set point remains unchanged. When the core temperature is set higher, fever occurs.

The action of the pre-optic region of the anterior hypothalamus in response to infection sets the core temperature. Hyperthermia, however, occurs when the body temperature rises without a change in the heat control centers. Watch this [video](#).

## Causes of Hyperthermia

### Failure of thermal homeostasis and increased heat production

- Exercise-associated hyperthermia:- a continuum of heat-related conditions due to [environmental](#) heat and exercise.
- Heat stress/cramps
- Heat exhaustion
- Heatstroke

- Seizures
- Agitation
- Uncoupling of oxidative phosphorylation- e.g., Salicylate overdose.
- Hepatic Metabolism stimulation-e.g - sympathomimetic drugs

## Hyperthermia secondary to other processes

- Neuroleptic Malignant Syndrome(NMS) – [link](#)
- Serotonin Toxicity/syndrome(SS) – Serotonin TOXICITY – [link](#)
- Malignant Hyperthermia (MH) – [link](#)

## Differential Diagnoses

There are multiple differentials to the cause of hyperthermia. A good history from the patient (if possible), carers or relatives is crucial to the diagnosis. Few important differentials to consider in ED are:

- Central nervous system infections,

- Status epilepticus,
- Stroke,
- Brain trauma,
- Neoplasms,
- Acute intermittent porphyria,
- Tetanus,
- Thyroid Storm
- Heat stroke,
- Sepsis.
- SSRI toxicity and other drug toxicities,
- pheochromocytoma.

## Malignant Hyperthermia (MH)

Incidence is about 1:10000-15000. All races are affected.

Mortality rates have fallen from 70-80 % to 2-3 % due to increase awareness, monitoring standards, and Dantrolene.

Genetically inherited disorder (autosomal dominant). About 70% of families are

linked to the RYR1 gene located on chromosome 19q. Triggering drugs cause a release of sarcoplasmic reticulum  $Ca^{2+}$ . Resulting  $Ca^{2+}$  stimulated glycolysis, muscle contraction, uncoupling of oxidative phosphorylation leading to hyperthermia. Drugs causing MH are volatile inhalation halogenated anesthetics and muscle relaxant suxamethonium.

## Pathophysiology

The usual body temperature of humans is between 36° C and 37.5°C.

When Core Body temperature (Rectal temperature/esophageal temperature) is greater than about 41.5°C it results in:

- Progressive denaturing of number of vital cellular proteins.
- Failure of vital energy-producing process in the cells like oxidative phosphorylation and failure of enzyme function.

- Loss of cell membrane function with increasing permeability.

#### **Tissues most at risk are:**

- Vascular endothelium
- Nervous tissue
- Hepatocytes

#### **At organ level hyperthermia manifests as :**

- Rhabdomyolysis
- Electrolyte disturbance
- Renal and Liver failure
- Cardiovascular dysfunction
- Acute Pulmonary edema with ARDS (acute respiratory distress syndrome)
- Disseminated intravascular coagulation (DIC)
- Neurological damage

#### **Clinical Features**

- Failure to achieve muscle relaxation following succinylcholine, e.g., master spasm impeding intubation and persisting for 2 minutes.
- Signs of increased metabolism: Tachycardia, Tachypnoea, dysrhythmia, increased CO<sub>2</sub> production noted on the end-tidal CO<sub>2</sub> monitor
- Metabolic acidosis, rhabdomyolysis and hyperthermia and DIC.
- Signs and symptoms may be delayed or may reappear after successful treatment.

### **Neuroleptic Malignant Syndrome(NMS) and Serotonin Toxicity/ syndrome(SS)**

Occurs due to increased motor activity and central resetting of hypothalamic thermostat

### **Neuroleptic Malignant Syndrome(NMS)**

There is Dopamine depletion/ dopamine receptor (D<sub>2</sub>) blockade in the hypothalamus, nigrostriatal pathways and spinal cord which leads to increased muscle rigidity and tremor via extrapyramidal pathways.

Hypothalamic D<sub>2</sub> blockade leads to elevated temperature set point and impairment of heat dissipation. This is an idiosyncratic reaction to neuroleptic agents. All classes of antipsychotics have been associated with neuroleptic malignant syndrome, most frequently in patients taking haloperidol and chlorpromazine. It occurs in response to a single agent and may occur in therapeutic dosages. It maybe dose-related and more commonly seen in patients on higher dose, depot neuroleptics.

#### **Clinical Features**

Usually develops in patients who have recently started a neuroleptic treatment or have recently increased the dose. The onset of the symptoms is 4-14 days after

the first day of therapy; most of the cases occur within 10 days. Usually associated with almost all antipsychotics and also in patients in whom dopaminergic agents have been withdrawn (e.g., in Parkinson's). Similar to Serotonin syndrome, there is a latent period of several hours to days.

#### Four Classic Signs

1. Hyperthermia  $>38^{\circ}\text{C}$
2. Severe Muscular rigidity (typically "lead pipe" rigidity)
3. Altered mental state
4. Autonomic instability

There is a broad spectrum of clinical illness;

Diaphoresis, Pallor, Dysphagia, Dyspnea, Tremor, Incontinence, shuffling gait, agitation, delirium progressing to lethargy, stupor, coma

**Examination findings showing autonomic dysregulation include:**

- Diaphoresis, sialorrhea, tachycardia, tachypnea, respiratory distress, hypertension / labile blood pressure, hypoxia.

Good drug history may help to differentiate between the NMS and Serotonin syndrome.

### Serotonin syndrome

There is excess CNS (Central Nervous System) Serotonin (5HT- 5Hydroxy Tryptamine) due to multiple mechanisms based on different 5HT receptors in the brain.

SS is most often caused by simultaneous ingestion of 2 or more serotonergic medications. There may be a recent history of dose increase. Excess serotonergic activity can be precipitated by any of the following mechanisms and drugs (ones in bold seen commonly in ED as overdose):

- Direct 5HT-receptor stimulation — Buspirone, triptans, lithium, carbamazepine, lysergic acid

diethylamide (LSD), mescaline-containing cacti (peyote and others).

- Direct 5HT release from stored vesicles — Amphetamines, MDMA, cocaine, reserpine, levodopa, MAOIs (monoamine oxidase inhibitors), codeine, dextromethorphan, pentazocine.
- Increased availability of 5HT precursors — L-tryptophan.
- Decreased 5HT reuptake — SSRIs (slow serotonin reuptake inhibitors), trazodone, nefazodone, venlafaxine, TCAs (Tricyclic antidepressants), dextromethorphan, tramadol, meperidine, (, Hypericum species (St. John's wort), amphetamines, carbamazepine, methadone, linezolid.
- Decreased 5HT degradation — MAOIs, St. John's wort.

### Clinical Features

CNS, Autonomic and Motor Dysfunction related features



- Agitation, anxiety, confusion, decreased level of consciousness, seizures
- Clonus, Hyperreflexia, Hypertonia, Incoordination, Myoclonus, Tremor
- Diaphoresis, Diarrhoea, Hypertension, Hyperthermia, Tachycardia
- mydriasis, piloerection, and muscular rigidity
- CVS features include sinus tachycardia, flushing, hypertension, and hypotension (rare).
- Citalopram causes dose-dependent QT prolongation.

Develops after a latent period, ranging from few hours to several days. Most patients are mildly affected, but the disease spectrum is very broad. Most cases resolve within 24-48 hours after withdrawal of the precipitating agent.

## Workup

Diagnosis of hyperthermia disorders is based on a detailed history, clinical

picture, and exclusion of alternative diagnoses. Drug history is very important and clinical suspicion is paramount. Investigations are directed towards the exclusion of other causes of pyrexia, e.g., sepsis and other disorders

Investigations are done to rule out complications and guide treatment.

- Serum electrolytes- to check imbalance and supportive treatment
- Creatinine Kinase – Guides treatment of Rhabdomyolysis.
- Serum Glucose – Rule out hypo/ hyperglycemia as the cause of altered mental state.
- ECG- arrhythmia, electrolyte abnormalities.
- Urine- Toxins
- Multiorgan dysfunction workup
  - Clotting screen- coagulopathy (DIC)LFT's, Renal functions- detect complications such as

AKI(Acute Kidney Injury) and rhabdomyolysis.

- Cardiac enzymes.
- Chest X-ray- To investigate complications and rule out the differential diagnosis.
- Specialist investigation:
  - Muscle biopsy using in-vitro contracture test (IVCT) which is the gold standard for MH diagnosis. This is done in specialized MH centers. 8 to 10 muscle specimens are taken and considered positive if muscle contracts to halothane and/or caffeine

## Treatment

### Supportive and cooling measures for hyperthermia in general

- Evaporative cooling- Remove all clothing, and spray the patient with tepid water while blowing air with a fan.

Areas with increased vascular beds, e.g., neck, axillae, groins should be asked with ice packs.

- Iced water immersion: – Can cause awkward patient access and difficulty in monitoring. Not very practical in the Emergency Department.
- Invasive methods: Cold IV fluids, urinary bladder lavage, peritoneal/pleural lavage with cold fluid.

## Serotonin Syndrome( SS)

- Mild cases- May need observation in ED for a few hours and safely discharged if asymptomatic.
- More serious cases would need supportive treatment and pharmacological therapy with observation and treatment for complications in ICU.
- Pharmacological Therapy
  - Mild SS- No treatment is needed or small doses of benzodiazepines.

- Severe SS: Neuromuscular paralysis should be considered early especially in cases with a low GCS.

- Antiserotonergic drugs:

- Chlorpromazine- 12.5-50mg IM/IV
- Cyproheptadine- 4-8mg orally 8 hourly.

## Neuroleptic Malignant Syndrome

- Benzodiazepines for anxiety and agitation.
- Stop all neuroleptics
- Correct volume depletion and hypotension with intravenous fluids
- Reduce hyperthermia (see above)
- Alkalinization of urine with sodium bicarbonate for prevention of renal failure following rhabdomyolysis.

- Bromocriptine as dopamine agonist can be given orally/NG tube, 2.5-10mg TDS.

## Malignant Hyperthermia

- Avoiding the triggering agents prevents MH.
- Using inhalation agent free machines during anesthesia.
- Dantrolene Sodium- Inhibits the release of calcium from the sarcoplasmic reticulum.

- 2.5mg /kg IV initially repeated every 15 minutes to maximum 30mg/ kg.

- AAGBI guidelines for treatment of MH poster [link](#)

## Prognosis and Disposition

- Early intensive care referral is indicated.
- Prognosis is worse with complications and multi-system failure.
- Mortality remains high in this group if untreated.

- Malignant hyperthermia: Modification of anesthesia in the future with inhalation agent free machines and no use of suxamethonium. Family members should be tested for susceptibility.

**References and Further Reading**, click [here](#)

# Selected Gastrointestinal Emergencies





# Acute Appendicitis

---

by Ozlem Dikme

## Case Presentation

*A previously healthy 22-year-old male was brought to the emergency department (ED) with recently-started **abdominal pain**. He had not eaten anything since that morning due to loss of appetite. He was nauseated and vomited three times. His abdominal pain started around the umbilicus and epigastric area. His pain increased as it moved towards his right lower quadrant (RLQ). The maximum pain was felt on the right iliac fossa. He had not taken any medication. His social history revealed that he was non-drinker, non-smoker and did not use any illicit drugs. His diet mostly consisted of carbohydrates. The past and family histories were unremarkable. His blood pressure was 120/70 mmHg, pulse rate was 100/min, the temperature was 37.8°C (100°F), and respiration rate was 22/min. Physical examination showed normal bowel sounds, tenderness and voluntary guarding,*



Audio is available [here](#)



*particularly over the right iliac fossa. The costa-vertebral angles were not tender. Oral intake was stopped, intravenous (IV) catheter was inserted, blood and urine tests were planned, and fluid therapy was started. The urinalysis was normal. White blood cell (WBC) count was 14,500 with 89% polymorphous and 11% lymphocytes. The ultrasonography (USG) showed a non-compressible tubular structure of 9 mm in diameter at RLQ. He admitted to the surgical ward with the diagnosis of acute appendicitis.*

Can you name the finding in the given ultrasound in this [video](#)?

## Introduction

About 7% of the population develops appendicitis in their lives. Males are affected 1.4 times higher than females, and teenagers more than adults (3:2). The incidence rises gradually from birth, peaks in the late teens, and declines in the elders. It occurs in all age groups but most frequently between the age of 10 and 30. Prevalence is higher in countries with diet habits low in fiber and high in refined carbohydrates. Low dietary fiber causes fecalith formation and obstruction of the appendicular lumen.

## Critical Bedside Actions and General Approach

The general approach to a patient with possible acute appendicitis must start with the patient stabilization. Fortunately, the most of the patients come with stable clinical presentation except pain. Some patients may present late. In this situation perforation is a possibility. Therefore

diffuse abdominal infection, systemic infection, even sepsis/septic shock can be another priority for the physician. After the ABC evaluation, focused gastrointestinal and pelvic orientation follows. Depending on the patient needs, critical actions necessary in the initial ABC evaluation can be applied. However, placing IV catheters, starting fluid therapy are the priority in most of the cases. Oral intake should be stopped. Pain medication and application of antibiotics may be considered in the early phase depending on the patient situation.

The possibility of acute appendicitis must be explained, and the patient's approval should be obtained for further steps. The evaluation should include laboratory tests and imaging. Count blood cell (CBC) and c-reactive protein (CRP) are generally not specific to diagnose, but they may be useful to confirm or exclude the differential diagnoses. USG or computed tomography is possible imaging modalities.

## History and Physical Examination Hints

Abdominal pain is the most common complaint. It typically starts periumbilical or epigastric, then migrates to the RLQ. It is the most discriminating feature of the patient's history. Its' sensitivity and specificity are approximately 80%; the positive likelihood ratio is 3.18, the negative likelihood ratio is 0.5. Patients typically avoid moving because it worsens their pain. The classic history of anorexia, periumbilical pain followed by nausea, RLQ pain, and vomiting occurs in only 50% of cases. Nausea is present in 61-92% of patients, anorexia in 74-78%. Vomiting almost always follows the pain. Diarrhea or constipation is noted in as many as 18% of patients. In up to 50% of cases, local tenderness of Mc Burney's point and rebound tenderness may be present. Typical physical findings are rebound tenderness, pain with percussion, guarding and rigidity. RLQ tenderness is seen in 96% of patients, but it is nonspecific. Other signs of

peritoneal irritation are triggered RLQ pain with palpation of the left lower quadrant (Rovsing sign), with internal and external rotation of the flexed right hip (Obturator sign), with the extension of the right hip (Psoas sign), with cough (Dunphy sign) or with dropping from standing on toes to the heels (Markle Sign).

## Differential Diagnoses

Many different specific diseases cause abdominal pain. The below list is given in alphabetical order. We advise you that look for other specific disease entity chapters to understand presentation, diagnosis and treatment differences.

- Acute Cholecystitis or Biliary Colic
- Acute Gastritis or Peptic Ulcer Disease
- Colonic carcinoma
- Diverticulitis
- Gastroenteritis

- Inflammatory Bowel Disease (Crohn Disease, Ulcerative Colitis)
- Inguinal hernia
- Intussusception
- Meckel Diverticulum
- Mesenteric adenitis
- Mesenteric ischemia
- Omental torsion
- Pancreatitis
- Perforated viscus
- Rectus sheath hematoma
- Tubo-ovarian pathologies (Ectopic pregnancy, Pelvic inflammatory disease, Abscess, Endometriosis, Ovarian cyst/torsion, Uterine leiomyomata)
- Typhoid Fever
- Ureterolithiasis or Urinary tract infection

## Emergency Diagnostic Tests and Interpretation

Appendicitis is a clinical diagnosis. However, some laboratory tests may help emergency physicians in the decision-making process. Each test has some pros and cons. Therefore, your clinical history and exam should be the main part of your decision-making process. Relying on laboratory tests may mislead you in some cases.

Count Blood Cell (CBC) is an easily accessible and inexpensive test, but it is nonspecific. Studies consistently show that WBC count is greater than 10500/mm<sup>3</sup> of 80-85% adult patients with acute appendicitis. Also, the neutrophil count is higher than 75% in 78% of patients. CBC shows different likelihood ratios (LR) for different WBC levels. LR of WBC of 9-11000 is 0.29. However, WBC of 11-13000 has 2.8 LR.

C-reactive protein (CRP) is useful, and it usually is higher than 1 mg/dL. However, it cannot detect the site of infection.

Therefore, it is not specific to appendicitis. Studies show that sensitivity of CRP is between 93% and 96.6% for acute appendicitis. A normal CRP level has a negative predictive value of 97-100% for appendicitis in the patients with symptoms longer than 24 hours. Investigators have also studied the combinations of WBC count, CRP and neutrophil count to reliably rule out the diagnosis of acute appendicitis. Patients with a WBC count below 10000/mm<sup>3</sup> and a CRP below 6 to 12 mg/dL are unlikely to have acute appendicitis (Negative likelihood ratio: 0.09). Patients with a WBC count above 10000/mm<sup>3</sup> and a CRP above 8 mg/dL were likely to have acute appendicitis (positive likelihood ratio: 23.32).

Urinalysis may differentiate diagnoses such as urinary tract infections. However, the appendix has a relationship with the right ureter, and in some cases, pyuria may not refer to only urinary infections. Pyuria may occur in cases of appendicitis, but severe pyuria marks

more likely urinary tract infections. Additionally, proteinuria and hematuria in urinalysis usually suggest genitourinary or hematological disorders. Women of childbearing age must have pregnancy evaluated. Ectopic pregnancy should be in your mind always.

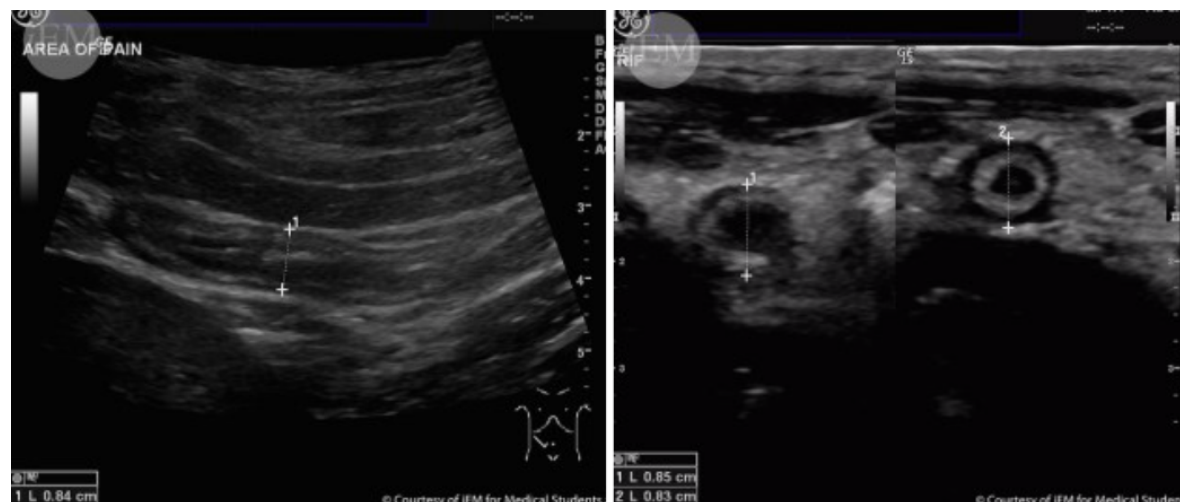
Computed tomography (CT) has 94% sensitivity and 95% specificity and shows higher diagnostic accuracy over USG (Sensitivity: 88%, specificity: 94%) for acute appendicitis. A large, single-center study found that CT has a high rate of sensitivity and specificity (98.5% and 98%, respectively) for acute appendicitis. Though the use of IV and oral contrast may increase sensitivity, it may prolong ED stays, cause allergic reactions and vomiting. Therefore, in adults, abdominal and pelvic CT may be performed with or without contrast.

A healthy appendix usually cannot be viewed by Ultrasonography (USG). In the case of acute appendicitis, the USG typically demonstrates a non-

compressible tubular structure of 7-9 mm in diameter in the RLQ. However, USG is not as accurate as CT. USG is the first choice, especially in pediatric patients, pregnant females, and slender patients. Additionally, if a gynecologic pathology is more likely than acute appendicitis in females, USG can be the initial test to detect gynecologic pathologies such as ectopic pregnancy, ovarian cysts or other female reproductive system pathologies. If the operator is an experienced ultrasonographer, it may be the first imaging method. It is also important to emphasize that USG is an operator-dependent modality.

Ultrasound images show the increased size of appendicitis (below). More than 6 mm is considered abnormal (Image 7.1).

**Image 7.1**



The ultrasound **video** shows transverse and longitudinal views of appendicitis in the same sequence. The appendix is located 3-4 cm deep from the skin surface. This video does not include measurement. However, the reported diameter was 8 mm, the diameter reaches more than 1 cm (10 mm) in some slices.

Plain radiographs are not specific or cost-effective. It may visualize an appendicolith (It is highly suggestive of appendicitis but only seen in fewer than 10% of patients) or air-fluid level on RLQ location.

If USG is equivocal, magnetic resonance imaging (MRI) should be considered in pregnant patients. Its' disadvantages are long scan times, high cost, and limited availability. Some researchers suggest MRI instead of USG in pediatric patients. MRI's (100%) sensitivity is found higher than USG (76%) in pediatric patients with acute appendicitis.

## Emergency Treatment Options

Very few patients require aggressive resuscitation during the initial evaluation (ABC phase). All patients with suspected dehydration or septicemia must receive IV access and aggressive crystalloid therapy. Additionally, parenteral antiemetics and analgesics should be administered. Prophylactic antibiotics should be given to cover gram-negative and anaerobic organisms.

One of the important global discussion is analgesic use in appendicitis. There is still disagreement between different



physician groups, especially emergency physicians and surgeons in some facilities. 2011 Cochrane review reported that “The use of analgesia for acute abdominal pain does not mask clinical findings, nor does it delay diagnosis.” However, only recommended analgesics are opioids in these patients.

## Pediatric, Geriatric, and Pregnant Patient Considerations

Appendicitis has relatively high misdiagnosis rates at both extremes of age. In children, the misdiagnosis rate is 25 - 30%. The most common misdiagnoses are gastroenteritis and respiratory tract infections. The early symptoms like loss of appetite or vomiting are non-specific. They may easily lead the physician to other diagnoses such as gastroenteritis, urinary or respiratory infections.

Ten percents of the appendectomies are performed in the elderly. Misdiagnosis rates are high in this age group too.

Elders initially relate their symptoms to their comorbidities. As a result, late presentation to ED may cause diagnostic delays. Additionally, ongoing drugs' side effects may mask their acute condition. Therefore, a late presentation or insignificance of symptoms should not dissuade the clinician from the diagnosis. The diagnostic delay relates to increased mortality and morbidity. The mortality rates range from 0.1% to 1% in children, and it rises above 20% in patients older than 70 years. Overall, the perforation rate varies from 16% to 40%. Younger children have a higher perforation rate between 50-85%. Diagnostic delays may increase perforation rates up to 55-70% in patients older than 50 years.

The appendicitis incidence in the pregnant remains unchanged compared to the general population, but the changes in the presentation may delay the diagnosis. During pregnancy, appendix replaces toward the right kidney and rises above the iliac crest at about 4.5 months of gestation. RLQ pain

and tenderness may occur in the first trimester, but RUQ or flank pain may dominate later. The symptoms are similar to the first-trimester pregnancy symptoms such as nausea, vomiting, and anorexia. The physicians should consider appendicitis if these symptoms reappear later in pregnancy. However, WBC count is not reliable in pregnancy because of the physiologic leucocytosis. Imaging modalities USG or MRI can use for the diagnosis.

## Decision Making

Clinical findings guide risk stratification. Risk stratification scores guide diagnostic modalities and disposition decisions such as discharge, observation or surgical consultation. The Alvarado score is a well-known classification for appendicitis (Table 7.1).



**Table 7.1** Alvarado Score In Acute Appendicitis

CATEGORY	EXPLANATION	SCORE
Symptoms	Migration	1
	Anorexia or acetone (in the urine)	1
	Nausea or vomiting	1
Signs	Tenderness in right lower quadrant	2
	Rebound pain	1
	Elevation of temperature ( $>37.3^{\circ}\text{C}$ measured orally)	1
Laboratory	Leukocytosis ( $>10,000/\text{mm}^3$ )	2
	Shift to the left ( $>75\%$ neutrophils)	1
Total Score		10

Adopted from Alvarado A. A practical score for the early diagnosis of acute appendicitis. *Ann Emerg Med.* 1986;15(5):557-564. Please read the original article for further information.

## Score

1-4 Appendicitis unlikely

5-6 Appendicitis possible

7-8 Appendicitis probable

9-10 Appendicitis very probable

## Disposition Decisions

Patients with minimal physical findings and a strong alternative diagnosis or previous multiple episodes of similar pain are considered low risk. In low-risk patients, the best course of action is advising on signs of appendicitis and arranging close follow-up in 12 to 24 hours. Discharged patients should start on a liquid diet and advance to solids when their symptoms improve. Patients with non-specific abdominal pain, who require significant doses of opiates should be considered for admission. Equivocal patients mostly consist of women of childbearing age, men, and children with atypical signs. They should be considered for diagnostic testing or active observation. Men and children with classic presentations are at high risk and gain little benefit from further imaging. Emergency physician should consult the patient with general surgeon without delay. Acute appendicitis is the most common reason for emergent abdominal surgery and appendectomy remains the only curative treatment. Antibiotic treatment without appendectomy may be sufficient therapy for

uncomplicated appendicitis, especially in the pediatric population.

**References and Further Reading,** click [here](#)

# Biliary Disease

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by Dan O'Brien

## Case Presentation

*A 35-year-old woman presents to the emergency department with right upper quadrant pain of two hours duration. She awoke several hours after eating a large meal. Based on increasing pain and nausea she presents for evaluation. She denies vomiting, fever or dysuria. Her past history is notable for diet-controlled type II diabetes, dyslipidemia, and essential hypertension. Her BMI is 33. Her only medication is lisinopril 10 mg daily. She has never had surgery. Her social history is unremarkable. She neither drinks alcohol nor uses tobacco. She has begun to diet and reports recent weight loss.*

*Her temperature is 37°C, blood pressure: 110/70 mmHg, pulse: 90 beats per minute. Physical exam reveals an overweight female in mild distress secondary to right upper quadrant pain. She cannot find a position of comfort and describes the pain as similar to labor pains. Pertinent exam*



Audio is available [here](#)

findings include: chest exam normal, cardiac exam normal, abdominal exam demonstrates normal bowel sounds and no rebound in any quadrant. She has guarding to inspiration with palpation over the gallbladder (positive Murphy's sign). Rectal exam normal, stool is hemoccult negative for blood. Pertinent lab values: glucose 110 mg/dl, alkaline phosphatase 120 U/L, alanine aminotransferase (ALT) 25 U/L, aspartate aminotransferase (AST) 25 U/L, gamma glutamyl transferase (GGT) 20 U/L, direct bilirubin 0.1 mg/dL, total bilirubin 0.5 mg/dL, lipase 20 U/L.

The emergency physician performs a focused right upper quadrant ultrasound (Image 7.2) and finds gallstones without associated gallbladder wall thickening or pericholecystic fluid. In addition, the patient has a "sonographic Murphy sign": there is maximal abdominal tenderness when the ultrasound probe is pressed over the visualized gallbladder.

Image 7.2



An IV was established, and the patient received an isotonic fluid bolus. In addition, ketorolac 30 mg IV and ondansetron 4 mg IV were administered. Over the course of an hour symptoms resolved. Absent evidence of gallbladder inflammation or infection, she was discharged from the emergency department and

*referred to a general surgeon for elective cholecystectomy. She was advised that her pain might return but if it is prolonged, is associated with fever or jaundice she is to return to the emergency department.*

## **Critical Bedside Actions and General Approach**

Abdominal pain is a common complaint in the emergency department and can be challenging to diagnose. Presenting illnesses may range from benign self-limited diseases to true surgical emergencies. The priority is to assess the stability of the patient. Use history, the likelihood of disease, vital signs, and the physical exam to assist in determining whether a patient may have a serious illness or surgical emergency. For example, a sudden onset of tearing pain radiating to the back in an older patient

with a history of hypertension may suggest a dissecting abdominal aortic aneurysm, but the colicky pain associated with abdominal distention may suggest bowel obstruction. Fever, protracted vomiting, syncope or gastrointestinal blood loss should all raise the suspicion of serious illness. In addition, it is important to exclude pregnancy and its complications in any woman of childbearing years who presents with abdominal pain. In this case, the patient had classic biliary colic and documented gallstones. The pain was due to crystals or a small stone passing and or blocking the cystic duct. Based on the resolution of pain, the absence of abnormalities on ultrasound exam such as pericholecystic fluid or wall thickening, and normal laboratory values it would be safe to discharge this patient for elective cholecystectomy. During the course of the management, it is very important to differentiate critical situations from uncomplicated gallbladder disease. In addition, the

physicians should think early pain medication to comfort the patient.

## **Differential Diagnosis**

Pain in the right upper quadrant can be of biliary origin including cholelithiasis: gallstones without inflammation, cholecystitis: inflammation or infection of the gallbladder wall, or cholangitis: inflammation or infection of the biliary ducts. Pancreatitis independent of, or as a consequence of gallstone obstruction of the common biliary duct, choledocholithiasis, may present in a similar fashion as well. Hepatitis, gastritis, dyspepsia, peptic ulcer disease are other potential gastrointestinal causes of right upper quadrant pain. Appendicitis, especially in pregnant patients may present with symptoms of right upper quadrant pain. Non-abdominal diseases such as pneumonia or pleurisy on the right lung may present with right upper quadrant pain.



## History and Physical Examination Hints

“A 35-year-old woman presents to the emergency department with right upper quadrant pain of two hours duration. She awoke several hours after eating a large meal. Based on increasing pain and nausea she presents for evaluation. She denies vomiting, fever or dysuria. Her past history is notable for diet-controlled type II diabetes, dyslipidemia, and essential hypertension. Her BMI is 33. Her only medication is lisinopril 10 mg daily. She has never had surgery. Her social history is unremarkable. She neither drinks alcohol nor uses tobacco. She has begun to diet and reports recent weight loss.”

Gallstones are two to three times more common in women, especially during childbearing years. The risk of also gallstones increases with age. Obesity or Body Mass Index (BMI) greater than 30 is associated with increased gallstone formation. Type II diabetes is associated with obesity, hyperlipidemia, and gallbladder hypomotility. Diabetic patients

are at increased risk for pancreatitis as well. Diets low in fiber and high in carbohydrates and fat have been associated with gallstone formation. This may, in part, explain regional differences in gallstone formation.

“Her temperature is 37°C, blood pressure: 110/70 mmHg, pulse: 90 beats per minute. Physical exam reveals an overweight female in mild distress secondary to right upper quadrant pain. She cannot find a position of comfort and describes the pain as similar to labor pains. Pertinent exam findings include: chest exam normal, cardiac exam normal, abdominal exam demonstrates normal bowel sounds and no rebound in any quadrant. She has guarding to inspiration with palpation over the gallbladder (positive Murphy’s sign). Rectal exam normal, stool is hemoccult negative for blood.”

Patients with biliary colic have moderate to severe right upper quadrant colicky pain without peritoneal signs. Although

described as colic, the pain may be more constant as it is caused by an obstruction of bile flow with subsequent distention. Patients may appear restless and unable to find a comfortable position. Murphy’s sign (the sudden cessation of a deep inspiration when the inflamed gallbladder descends and reaches the examiners’ fingers palpating the right subcostal area) is 65% sensitive and 87% specific for acute cholecystitis. Fever is not typical, and jaundice is rarely seen unless there is obstruction of the common bile duct from choledocholithiasis or extrinsic compression due to mass or inflammation.

## Emergency and Diagnostic Tests and Interpretations

### Laboratory Tests

“Pertinent lab values: glucose 110 mg/dL, alkaline phosphatase 120 U/L, alanine aminotransferase (ALT) 25 U/L, aspartate aminotransferase (AST) 25 U/L, gamma glutamyl transferase (GGT) 20 U/L, direct

bilirubin 0.1 mg/dL, total bilirubin 0.5 mg/dL, lipase 20 U/L.”

Alkaline phosphatase (ALP) is synthesized by the bile duct epithelial cells. Its production is stimulated by bile duct obstruction and is elevated in a majority of patients with cholestasis. However, isoenzymes are found in the liver, bone, placenta, small bowel and leukocytes; it is therefore not specific for the biliary tract.

Bilirubin is a breakdown product of heme. Unconjugated bilirubin is hydrophobic and is transported in the blood bound to albumin. It is taken up by the hepatocyte, conjugated, and actively secreted into the biliary tract. Cholestasis may elevate serum bilirubin.

The aminotransferases; aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are found in the liver, cardiac and skeletal muscle, and cerebral nerve cells. Levels of these enzymes are typically only mildly elevated but may be markedly increased in

cholangitis. ALT may briefly spike during acute obstruction, but it usually is not elevated unless there is secondary liver parenchymal damage. An AST level greater than the ALT level suggests alcoholic liver disease, cirrhosis or metastatic disease.

Gamma-glutamyl transpeptidase (GGT) is a membrane-bound peptidase that hydrolyzes peptides to amino acids and smaller peptides. Although serum activity is primarily from the liver, it is found in the renal proximal tubule, pancreas, and intestine. Its circulating half-life is usually 7-10 days but may increase to 28 days in alcohol-associated liver disease. The cholestatic disease may elevate GGT significantly. A complete white blood cell count, serum electrolytes, glucose renal function studies, and urinalysis, may assist in diagnosis and management.

## Imaging Modalities

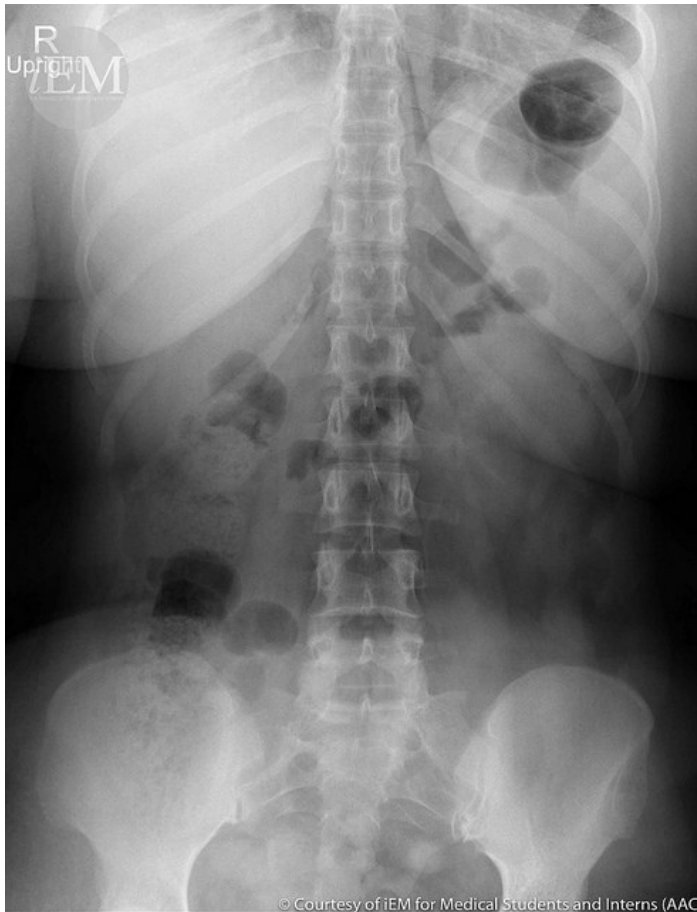
“The emergency physician performs a focused right upper quadrant ultrasound and finds gallstones without associated

gallbladder wall thickening or pericholecystic fluid. In addition, the patient has a “sonographic Murphy sign”: there is maximal abdominal tenderness when the ultrasound probe is pressed over the visualized gallbladder.”

Plain radiography is often not helpful in assessing biliary stones as most do not contain enough calcium to be visible on plain x-ray. Plain imaging may be useful to identify gas in the biliary tree or evidence of intestinal obstruction.

X-ray (Image 7.3) shows relatively normal findings in a RUQ and abdominal pain patient. The CT scan of the same patient is shown below. It revealed cholecystitis ([Image 7.5](#)).

**Image 7.3**



- pericholecystic fluid,
- sonographic Murphy's sign,
- common duct dilatation.

In acute cholecystitis, gallstones are present in 95-99% of cases. Emergency physicians, performing focused, limited bedside ultrasound and taking into account the context of the patient's history and clinical picture have documented a sensitivity of 90-96%, a specificity of 88-96% as well as a positive predictive value of 88-99% and a negative predictive value of 73-96% for cholecystitis. (Image 7.4)

Ultrasound imaging of the right upper quadrant is the principal study used to evaluate biliary-type pain and detect gallbladder disease and biliary dilatation.

There are several sonographic criteria for acute cholecystitis;

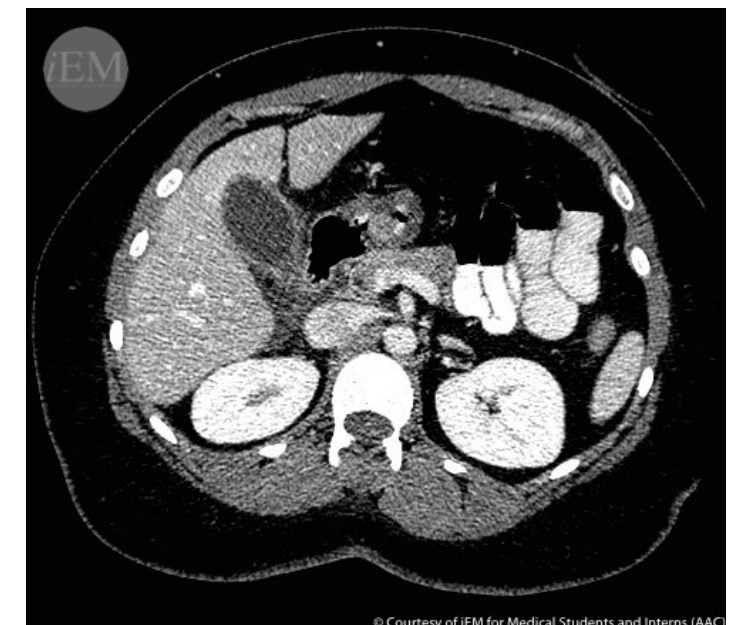
- the presence of gallstones,
- thickened gallbladder wall,

**Image 7.4**



C T imaging (Image 7.5) is not nearly as helpful as the right upper quadrant ultrasound in evaluating the biliary system for evidence of cholecystitis. Gallstone sensitivity is about 75%, and common duct stones may be missed. It may be helpful to reveal complications of cholecystitis such as gangrenous or emphysematous cholecystitis as well as to exclude other pathologies in the abdomen.

**Image 7.5**





## Emergency Treatment Options

“An IV was established, and the patient received an isotonic fluid bolus. In addition ketorolac, 30 mg IV and ondansetron 4 mg IV were administered. Over the course of an hour symptoms resolved. Absent evidence of gallbladder inflammation or infection she was discharged from the emergency department and referred to a general surgeon for elective cholecystectomy. She was advised that her pain might return but if it is prolonged, is associated with fever or jaundice she is to return to the emergency department.”

Asymptomatic gallstones do not require any treatment. Most remain asymptomatic for years after diagnosis. About 1-2% may become symptomatic annually.

Biliary colic or biliary pain typically has a definitive onset with a duration ranging from 15 minutes to up to four hours. Antiemetics and nonsteroidal anti-

inflammatory drugs (NSAIDs) are first-line therapies. In fact, studies suggest that NSAIDs have similar efficacy as opioids with fewer complications. Opioids may be used to control pain. Although there were historical concerns about morphine causing greater sphincter of Oddi spasm relative to other opioids, all opioids to some degree increase sphincter of Oddi pressure and biliary pressure. If a patient's pain is resolving and controlled with oral agents, they may be discharged and referred to a general surgeon for consideration of elective laparoscopic cholecystectomy.

Acute cholecystitis is best managed in the hospital with surgical consultation. Early laparoscopic cholecystectomy is often the treatment of choice. Patients should be given nothing by mouth. About 20% of patients develop gallbladder or biliary duct infection. Appropriate antibiotics regimens include second- and third-generation cephalosporins, carbapenems,  $\beta$ -lactam/ $\beta$ -lactamase inhibitor combinations or a combination

of metronidazole and a fluoroquinolone. Most patients will improve over 24 to 72 hours before surgical intervention.

Cholangitis, an infection of the bile duct, is a life-threatening disease that requires aggressive resuscitation, timely antibiotics, and early drainage via either endoscopic retrograde cholangiopancreatography (ERCP) guided sphincterotomy or stent placement or percutaneous drainage to stabilize the patient prior to definitive surgery.

## Pediatric, Geriatric, Pregnant Patient and Other Considerations

None other than mentioned above.

## Disposition Decisions

### Admission

Patients with suspected cholecystitis or cholangitis should be admitted to the hospital. For suspected cholangitis, emergency consultation, and if need be, transfer to a facility that can emergently

establish biliary drainage either via ERCP-guided sphincterotomy or percutaneous stenting.

## Discharge

Patients with biliary colic may be discharged once their symptoms have resolved with follow up with a general surgeon. They should be informed that there may be symptom recurrence and should be instructed to return if they experience prolonged pain, fever or jaundice.

## Referral

Asymptomatic gallstones need not be referred to a general surgeon. The patient should be informed of their findings and instructed to follow up with their primary care physician.

**References and Further Reading**, click [here](#)



# Massive Gastrointestinal Bleeding

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by Dan O'Brien

## Case Presentation

*A 68-year-old female presents to the emergency room at midnight, with a chief complaint of vomiting “coffee grounds” earlier that evening. She has a history of congestive heart failure, hypertension, and a mild stroke. Her medications include lisinopril 20 mg, Lasix 20 mg, aspirin 325 mg, and clopidogrel 75 mg daily. Recently, she has taken ibuprofen several times daily for arthritic pains. Family history is significant for peptic ulcer in her mother and a brother. Pertinent Exam: blood pressure is 98/65 mmHg, heart rate 110 bpm and regular, respiratory rate 14, non-labored, and temperature 37 °C. She appears pale, has a poor capillary refill and is mildly confused but oriented to person, place and time. Heart and lung sounds are normal; her abdomen is soft, non-tender, without organomegaly, and without bruits. She has trace pedal and pretibial edema. Her neurological exam is*



Audio is available [here](#)

*grossly normal. While being examined, she asks for a bedpan and vomits a cup full of bright red blood. Her blood pressure systolic is now 85 mm Hg.*

*She has hemodynamically significant upper GI bleeding. Her hypotension and tachycardia indicate loss of more than 20% total blood volume. The most likely working diagnosis is active upper GI bleeding likely from peptic ulceration secondary to nonsteroidal anti-inflammatory drugs (NSAIDs) with likely *Helicobacter* infection.*

*Bedside testing reveals hemoglobin of 6 g/dL. Transfusion with packed red blood cells is begun with a goal of hemoglobin of 8.0-10.0 g/dL. Additional labs demonstrate a normal albumin and prothrombin time (PT). Her creatinine is 2.0 mg/dL. Old records document baseline*

*hemoglobin of 11 g/ dL and creatine of 1.0 mg/ dL.*

*Initial resuscitation is successful. Upper endoscopy reveals a bleeding duodenal ulcer that is successfully stopped with hemoclips. The patient's *H. pylori* stool antigen is positive. Cardiology and Neurology agree to stop aspirin and clopidogrel. Oral iron was started, and the patient was discharged. With the avoidance of NSAIDs and with *H. pylori* eradication the risk of rebleed is less than 5%.*

## Introduction

Despite advances in diagnosis prevention and treatment, nonvariceal upper gastrointestinal bleeding is still a serious problem in clinical practice. The incidence ranges from 48 to 160 cases per 100,000 population per year. Upper GI bleeding causes mortality ranges from 5% to 14%.

## Critical Bedside Actions and General Approach

*“She has hemodynamically significant upper GI bleeding.”*

### Indicators of Major Blood Loss

- Acidosis
- Anticoagulation
- Antiplatelet medications
- Azotemia (BUN > 40 mg/dL)
- Chest pain or dyspnea
- Continued bleeding or re-bleeding
- End stage renal disease

- Hematochezia from upper GI source
- Hemoglobin <8 g/dL
- Liver cirrhosis, coagulopathy
- Orthostatic
- Resting tachycardia (>100 bpm)
- Syncope (systolic < 90 mmHg)
- Transfusion > 1 unit/8hrs or 6 units total

Initiate resuscitation: insert two large bore intravenous catheters infuse lactated ringers, type and crossmatch, obtain complete blood count with platelets, PT, and INR, as well as routine blood chemistries to assess for renal and hepatic function. Start intravenous octreotide, a somatostatin analog, at 50 mcg/hour. Proton pump inhibitor by continuous drip, as a pH of 7 or greater is needed for platelet function and clot adherence.

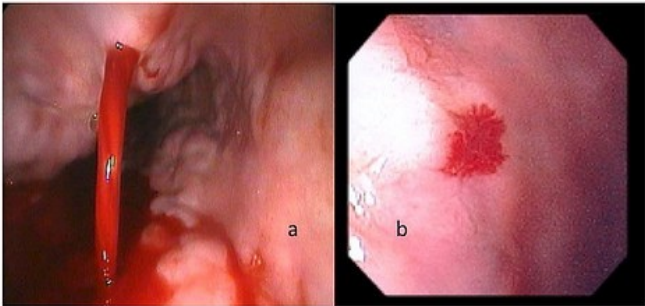
Intravenous erythromycin 250 mg if given within 30 minutes of planned endoscopy can improve visualization. If there is a

history of liver cirrhosis also give one gram IV Ceftriaxone or 400 mg norfloxacin orally twice daily. Antibiotics are of benefit in cirrhosis by decreasing infectious sequelae as well as the incidence of encephalopathy. The reduction of bacterial products in the portal circulation results in less vasodilation, which lowers the rebleeding risk. After initial stabilization efforts, consult an endoscopist.

*“The most likely working diagnosis is active upper GI bleeding likely from peptic ulceration secondary to nonsteroidal anti-inflammatory drugs (NSAIDs) with likely Helicobacter infection.”*

Figure 7.1

Figure 1. Pathophysiology of bleeding is related to vessel size and pressure. Arterioles or venules greater than 1mm can bleed as much as 300mls/hour. Image (a) shows an actively bleeding esophageal varix which requires urgent intervention. Image (b) shows a mucosal vascular ectasia, which may bleed overtly or below the detection limit of tagged cell imaging, <0.5 mls/hour. Endoscopy is more often diagnostic, with therapy applied for active bleeding.



Courtesy of Dan O'Brien

## Differential Diagnosis

While it is important to know what is bleeding to determine prognosis and guide management, it is most crucial to think of anatomy and pathophysiology: larger vessels bleed faster and more often require urgent intervention. The internal diameter and pressure in vessels above the ligament of Treitz are greater than vessels associated with lower GI bleeding.

Figure 7.2

Figure 2. *Helicobacter pylori* are spiral shaped bacteria that produce urease and insight a chronic inflammatory reaction that can lead to erosive gastritis, duodenal and gastric ulcers, gastric lymphoma and carcinoma. All patients with bleeding peptic ulcer should be checked for *H. pylori* infection, treated and have confirmation of eradication. Electron micrograph (a) and organisms found on gastric biopsy (b)



Courtesy of Dan O'Brien

Table 7.2 Comparison of Upper and Lower GI Bleeding

UPPER GI BLEEDING	LOWER GI BLEEDING
35% present with shock	19% present with shock
65% require transfusion	36% require transfusion
30% require intervention to stop	>90% stop spontaneously

The goal of therapy is to stop bleeding to prevent end organ ischemic damage. Medical treatment alone is successful for most cases of lower GI bleeding with a third of upper GI bleeding cases requiring emergent endoscopic therapy.

Table 7.3 Presentations of Upper and Lower GI Bleeding

UPPER GI BLEEDING	LOWER GI BLEEDING
Abdominal pain may or may not be present in peptic ulcer	Large volume hematochezia or maroon stool with orthostasis indicates bleeding from right sided diverticulae or arteriovenous malformations
Chest pain with esophageal ulcer	Small volume hematochezia without orthostasis indicates hemorrhoidal bleeding (usually painless) if painful with dyschezia indicates anal fissures or proctitis
Sudden fullness with nausea due to blood in GI tract	Bloody loose stools with low abdominal pain present in infectious colitis, inflammatory bowel disease or ischemic colitis
Hematemesis or coffee ground emesis followed by melena	
Hematochezia in 10% of rapidly bleeding upper lesions	
Coughing followed by hematemesis in Mallory Weiss tear	
Valsalva may prompt bleeding from esophageal or gastric varices	

Historical presentations vary in upper and lower GI bleeding



Upper GI bleeding represent 65 to 80% of all GI bleeding, and includes

- esophageal or gastric varices,
- duodenal or gastric ulcer,
- erosive gastritis,
- erosive or ulcerative esophagitis,
- Mallory Weiss tears,
- gastrointestinal cancers,

Rarer causes are

- hemobilia,
- splenic artery pseudoaneurysms,
- Dieulafoy lesions,
- gastrin-secreting tumors (Zollinger-Ellison syndrome),
- arteriovenous fistulae,
- penetrating foreign bodies,

- gastric antral vascular ectasias (GAVE or “watermelon stomach”) or arteriovenous
- malformations above the ligament of Treitz.

Worldwide, upper GI bleeding from peptic ulcer is most prevalent, although persons with portal hypertension may represent the majority who present with massive upper GI bleeding. With advanced age and atherosclerotic disease, more patients are using anticoagulants or antiplatelet medications that impair clot formation, and augment bleeding. Gastrointestinal bleeding from non-steroidal induced peptic ulcers is on the rise, with up to 1 in 2 adults taking these medicines. Although with industrialization and improved hygiene the prevalence of *Helicobacter pylori* has declined, the infection and associated conditions are still major causes of upper GI bleeding in many parts of the world including the Middle East, Asia, and South America.

Lower GI bleeding is most often caused by right-sided diverticula, arteriovenous malformations, colonic adenocarcinoma, ischemic colitis, inflammatory bowel disease, infectious colitis, or anorectal lesions including hemorrhoids, fissures, and proctitis.

## History and Physical Examination Hints

Elderly patients and those with valvular heart disease or renal failure have an increased risk for arteriovenous malformations anywhere in the GI tract. Chronic NSAID use causes gastric mucosal erosions in at least one-third of daily users or significant ulceration in 2%. Alcohol consumption, chronic viral hepatitis, non-alcoholic hepatitis (NASH) can result in cirrhosis with portal hypertension. A family or prior history of peptic ulcer suggests *Helicobacter pylori* infection. *H. pylori* is a spiral-shaped flagellated bacterium that lives in the human stomach and interrupts the protective mucous bicarbonate layer, thus exposing the epithelium to hydrochloric



acid leading to chronic inflammation. Most persons with duodenal ulcer report sharp epigastric pain worsened by eating while less than 50% of patients with gastric ulcer report abdominal pain that improves post meals, as the acid is then buffered by the food and duodenal bicarbonate secretion. The color of vomitus or stool is also predictive of severity: hematemesis suggests ongoing bleeding, whereas “coffee grounds” indicate partially digested hematin or “old blood.” Abrupt symptom onset associated with hypotension suggests acute bleeding whereas a history of weeks of intermittent dark melanic stools suggests chronic blood loss. Presenting vitals signs are most predictive of the magnitude of blood loss; other important physical clues for portal hypertension include abdominal ascites, enlarged liver or splenomegaly. Evidence of hyperestrogenemia in males with cirrhosis includes gynecomastia, testicular atrophy, and spider telangiectasias on the chest or upper body. Palmer erythema and bounding pulses in the fingers from peripheral vasodilation are indicators of advanced cirrhosis. The bedside physical exam is unreliable in females as palmer erythema and telangiectasias are normal findings. Ascites determination is difficult in every patient unless it is massive. If available, a bedside ultrasound may confirm suspected ascites, coarse echotexture of the liver, or show reduced or reversed (hepatopedal) flow in the hepatic veins in advanced cirrhosis.

Image 7.6



## Emergency Diagnostic Tests and Interpretation

When abdominal pain is present, a plain film with upright chest x-ray may reveal significant atherosclerotic disease, ingested foreign bodies, or subdiaphragmatic free air from a perforated ulcer.

*“Bedside testing reveals hemoglobin of 6 g/dL. Transfusion with packed red blood cells is begun*

*with a goal of hemoglobin of 8.0-10.0 g/dL. Additional labs demonstrate normal albumin and PT. Her creatinine is 2.0 mg/dL. Old records document baseline hemoglobin of 11 g/dL and creatine of 1.0 mg/dL.”*

Patients with low albumin, prolonged PT or INR, or platelet count less than 150K, consider underlying cirrhosis and avoid transfusion above hemoglobin of 7.5 to 8.0, to avoid increasing portal pressure and increasing the risk of rebleeding. A platelet transfusion would be indicated for a critically low platelet count (<50K) from consumptive coagulopathy or splenic sequestration. For significant coagulopathy, (INR >1.8) transfusing fresh frozen plasma (10-15 cc/kg) corrects factor seven deficiency to allow coagulation to occur. Cryoprecipitate or other specific factors may be indicated for patients with known factor deficiencies. Red blood cell indices are useful as a low mean corpuscular volume (MCV) suggests iron deficiency from chronic GI blood loss whereas in acute bleeding MCV is often increased due to the release of reticulocytes from the bone marrow.

## Emergency Treatment Options

*“Initial resuscitation is successful.”*

**Table 7.4** Emergency Management of GI Bleeding

TREATMENT OPTIONS	COMMENTS
Resuscitation with packed red blood cells	Goal: Hgb 7.5 to 8.0 in those with portal HTN  Hgb 9.5 to 10 in the elderly, MI, CHF, stroke
IV octreotide	50mcg/hour for active bleeding anywhere in the GI tract (both upper and lower bleeding)
IV continuous PPI for all upper GI bleeders	give IV erythromycin 250mg 30 minutes prior to EGD to improve visualization
Antibiotics	ceftriaxone 1 gram IV or norfloxacin 400mg IV twice daily for all GI bleeders with cirrhosis
Timing of endoscopy	It depends on magnitude of bleeding; goal is to stop bleeding as soon as possible to prevent end organ ischemic damage. Involve consultants early!
Other	Less than 5% cases require angiography, less than 1% surgery

It is not necessary to insert a nasogastric tube (NG) as the information obtained rarely changes management and may compound problems by causing pain, gagging, and epistaxis. A negative aspirate does not exclude active bleeding, and a positive

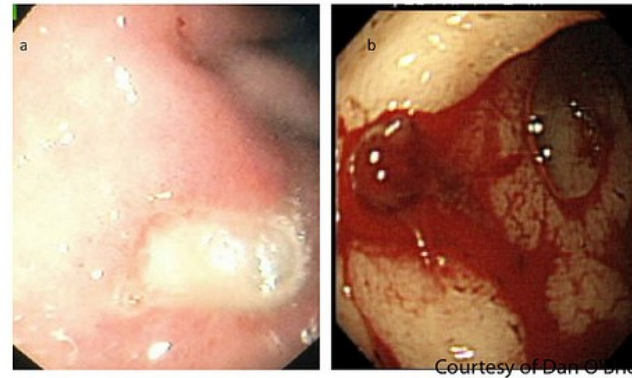
aspirate does not affect the timing of endoscopy or additional interventions. Gastric lavage is no longer considered useful. Consider endotracheal intubation to decrease aspiration risk before elective endoscopy for any patient with upper GI bleeding who is unconscious, in significant respiratory distress, or with recurrent witnessed hematemesis.

**Urgent Endoscopy:** Call as soon as possible for endoscopy in patients with hemodynamically significant bleeding. Endoscopy is portable and can be safely performed in the emergency department providing immediate information regarding diagnosis, treatment, prognosis, and disposition.

Large studies have shown that endoscopy can safely be performed in patients with bleeding leading to acute myocardial ischemia or infarction, with improved outcomes for interventions that stop further bleeding. The magnitude of bleeding, signs of continued bleeding, or suspicion of varices predicate the timing

**Figure 7.3**

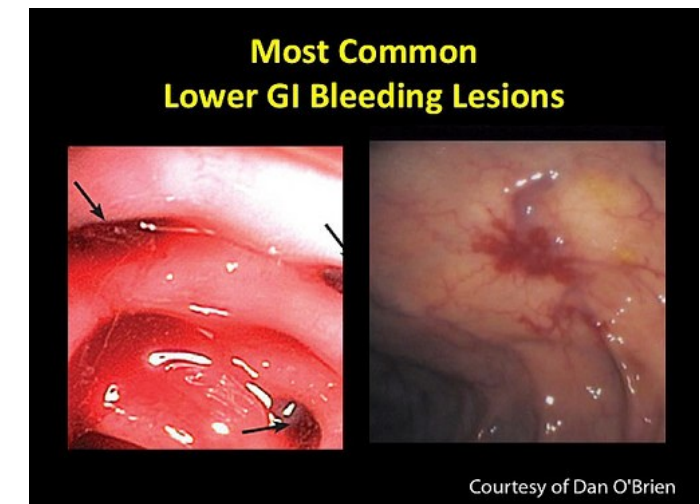
Figure 3. Prognosis is related to endoscopic findings: Patients with clean based ulcers (a) may be discharged home, whereas those with active bleeding (b) have a re-bleeding rate of 50% within 72 hours. They require ICU admission, therapeutic endoscopy, IV octreotide and IV continuous proton pump inhibitor to ensure platelet function and clot adherence.



of endoscopy in upper GI bleeding. Outcomes for endoscopic intervention have shown reduced rebleeding and transfusion requirements, with improved morbidity. With excellent clinical care and combined with endoscopic therapies, mortality from non-variceal GI bleeding is 10%, and mortality from variceal hemorrhage is 25-30%. Colonoscopy for diagnosis of lower GI bleeding cause is most often performed electively after resuscitation; usually within 24 to 48 hours. Visualization of the lower GI tract requires cleansing with large volume balanced electrolyte solutions taken orally or by NG for quicker delivery.

Colonoscopy is used less often as an interventional therapeutic technique to stop bleeding from hemorrhoids, fissures, arteriovenous malformations or diverticuli.

**Figure 7.4**



Interventional Radiographic Techniques are required in less than 5% of all cases of non-variceal GI bleeding. Angiography can arrest bleeding from arteriovenous malformations of the upper or lower GI tract, as well as selective embolization of arterioles from bleeding tumors or ulcers. Early use of transjugular intrahepatic portosystemic shunts (TIPS) to definitively reduce portal pressure is beneficial after initial endotherapy, and as first-line

therapy for select patients with high risk of variceal bleeding.

Emergency Surgery for GI bleeding is required in less than 1% of all cases including surgery for a peptic ulcer, and total or subtotal colectomy for shock associated with bleeding diverticuli. Emergency shunt surgery for liver cirrhosis is almost never performed, as the mortality is unacceptably high compared to endoscopic and angiographic techniques.

### Pregnant Patients and Other Considerations

In pregnant patients with GI bleeding, monitor for fetal distress, and consult Obstetrics. Avoid erythromycin in the third trimester otherwise treat the same as any other adult with GI bleeding. Emergency upper endoscopy is safe in all trimesters. Lower endoscopy may be difficult depending on the size of the fetus/uterus but is not contraindicated. The endoscopist will use safe sedation medications for pregnancy. For GI bleeding in patients with acute MI, significant heart disease, stroke or significant neurovascular disease consult cardiology or neurology for help with management. Often they will agree to urgent endoscopy to clarify and treat bleeding lesions without interruption or reversal of anticoagulation.

### Disposition Decisions

There are several valuable bleeding scoring systems help to guide disposition.

**Table 7.5** Glasgow-Blatchford Risk Score

CATEGORY	SCORE
<b>BUN in mg/dL</b>	
18.2 to 22.4	2
22.5 to 28	3
28.1 to 70	4
70.1 or greater	6
<b>Hemoglobin, men g/dL</b>	
12 to 13	1
10 to 11.9	3
9.9 or less	6
<b>Hemoglobin, women g/dL</b>	
10 to 12	1
9.9 or less	6
<b>Systolic Blood Pressure, mmHg</b>	
100-109	1
90-99	2
<90	3
<b>Heartrate &gt;100 peats per minute</b>	1
<b>Melena</b>	1
<b>Syncope</b>	2
<b>Hepatic Diseases</b>	2
Heart failure	2



Glasgow-Blatchford Risk Score is useful for predictive of inpatient mortality, blood transfusions, re-bleeding, ICU monitoring, and hospital length of stay. Patients with a score of zero may be discharged home, those with score 2 or higher are usually admitted, and those with score of 10 or more are at highest risk for morbidity and resource utilization. Maximum score is 23.

**Table 7.6** AIM65 Bleeding Score

RISK FACTOR	SCORE
Albumin <3.0	1
INR > 1.5	1
Altered mental status	1
SPB < 90mm Hg	1
Age > 65	1
MAXIMUM SCORE	5
Point	Mortality %
0	0%
1	0.9%
2	7.4%
3	42%
4	75%
5	100%

AIM65 GI Bleeding Score is practical, easy to remember, assists with level of care, and timing for endoscopy. Scores less than 1 predict good outcome, scores above 2 require hospitalization and treatment.

### Admission Criteria

Patients with GI bleeding presenting in shock, requiring transfusion or with bleeding scores (AIM65>2 or Glasgow-Blatchford (GB) >10) have significant predictable morbidity and mortality requiring ICU admission and treatment. Patients with AIM65 of 1 or less or GB score 2 or less have predictably mild GI bleeding (melena without hematemesis and who are hemodynamically stable) and may be cautiously admitted to a medical floor.

### Discharge Criteria

Patients with AIM or GB score of zero may be discharged home without outpatient gastroenterology evaluation within two weeks. Prescribe twice daily PPI, avoidance of NSAIDS and alcohol for those with upper GI symptoms. Instruct them to return immediately for syncope or signs of bleeding. Those with history and findings consistent with minor lower GI bleeding and stable hemoglobin should also be referred for outpatient consultation within 2 weeks.

“Upper endoscopy reveals a bleeding duodenal ulcer that is successfully stopped with hemoclips. The patients H. pylori stool antigen is positive. Cardiology and Neurology agree to stop



aspirin and clopidogrel. Oral iron was started, and the patient was discharged. With the avoidance of NSAIDs and with H. pylori eradication the risk of re-bleed is less than 5%.

**References and Further Reading**, click [here](#)

# Acute Mesenteric Ischemia

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by Rabind Antony Charles

## Case Presentation

*A 75-year-old woman presents to your Emergency Department (ED) with diffuse abdominal pain for the past day, associated with diarrhea and vomiting. She says the pain is increasingly worse and has failed to respond to paracetamol and charcoal tablets. She has a history of hypertension, hyperlipidemia, and atrial fibrillation. She has no history of laparotomy. She is alert and oriented. However, she is in distress because of her abdominal pain. The pain score is 9 out of 10. Blood pressure: 96 over 56 mmHg, pulse rate: 125 (irregularly, irregular), respiratory rate 20, pulse oximetry: 98% on room air, tympanic temperature: 37.5 degrees Celsius. Heart sounds: (irregular) S1S2 positive. Lungs sounds are bilateral equal and clear. Abdominal exam reveals diffuse tenderness; it is worse in periumbilical region, no guarding,*

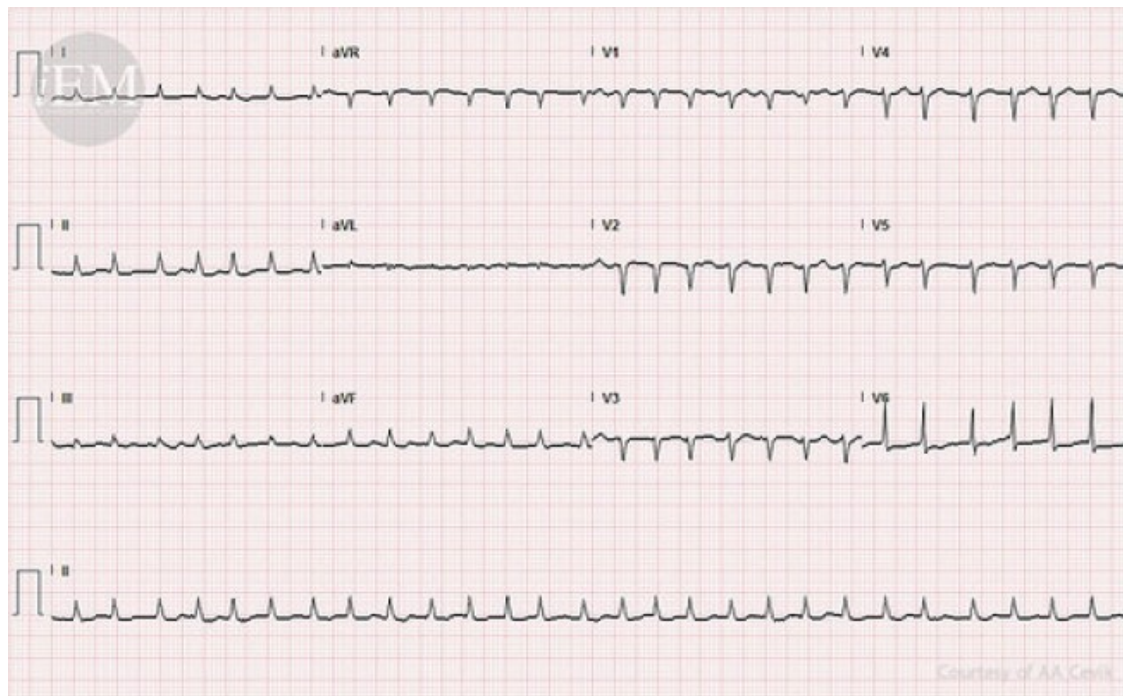


Audio is available [here](#)

*bowel sounds are sluggish. No scars or hernias noted. Per rectal exam: brown stool.*

What do you think about patient's ECG?

**Image 7.7**



## Introduction

Acute mesenteric ischemia is a life-threatening cause of **acute abdominal pain** which occurs predominantly in patients over 50 years old with the underlying cardiovascular disease. It is caused by inadequate flow through the mesenteric vessels resulting in bowel ischemia and eventually gangrene of the bowel wall. Mortality rates can be between 60-80% especially in patients with greater than a 24-hour delay in diagnosis or presentation. This underscores the importance of early detection in the ED, and the need for aggressive management to reduce morbidity and mortality. Surgical intervention in 6 hours of symptoms increases survival rate.

Mesenteric artery embolism is responsible for 50% of patients. Symptoms are sudden onset of abdominal pain with bloody diarrhoea if infarct develops. Arrhythmias (e.g., Atrial fibrillation), valvular disorders and recent myocardial infarction are the predisposing factors for embolism.

Mesenteric artery thrombosis is around 25% of the patients.

Patients are generally shown atherosclerotic disease symptoms.

“Abdominal angina” for preceding months, which is a pain on eating with loss of weight, then sudden severe pain episodes.

In 20% of the reason is non-occlusive mesenteric ischemia. Hypotension, Congestive Heart failure, dialysis, use of vasoconstrictors or digoxin are predisposing factors. This

situation happens typically intubated, sick ICU patients on vasopressors who deteriorate with bloody diarrhea and worsening hypotension.

If the patient has hypercoagulable states or his story of prior thromboembolic events, mesenteric venous thrombosis should be considered (5% of the patients). Patients present with nonspecific abdominal pain with diarrhea 1-2 weeks after the event which may resolve spontaneously.

## History Taking and Physical Examination Hints

This is a difficult condition to diagnose, especially in the early stages. However, it should be considered in those over 50 years old with severe abdominal pain, and predisposing factors for the condition.

In the early stages, patients may present with severe poorly localized abdominal pain, nausea, vomiting, and diarrhea with no signs of peritonism. These symptoms may mislead physicians into assuming

the patients are suffering from gastroenteritis.

One of the key features to look out for is pain that is “out of proportion” to the abdominal findings. This is due to visceral ischemia with sparing of the parietal peritoneum in the initial stages. Peritonitis is a late finding and points towards severe bowel ischemia and necrosis. At this stage, there may be abdominal distension associated with reduced bowel sounds.

Mesenteric ischemia also can be more subacute in its presentation with the insidious onset of less severe and vague abdominal pain, abdominal distension, and occult gastrointestinal bleeding.

## Differential Diagnoses

Depending on the clinical presentation, the differential diagnoses can be quite broad and include the following:

- Acute gastroenteritis
- Acute cholecystitis

• Acute pancreatitis

- Peptic ulcer disease
- Bowel perforation
- Diverticulitis
- Bowel obstruction
- Ureteric calculi

## Emergency Diagnostic Tests and Interpretation

### Laboratory Tests

There are “no” sufficiently sensitive or specific serum markers to identify acute mesenteric ischemia.

Complete Blood Cell Count: may show haemoconcentration and leukocytosis ( WBC count > 15,000/mm<sup>3</sup>) – lacks specificity.

Arterial/Venous blood gas analysis: metabolic acidosis is seen late in the disease. Presence of metabolic acidosis which cannot otherwise be explained



should prompt the clinician to suspect mesenteric ischemia in the appropriate clinical context.

Serum lactate: nearly 100% sensitive when bowel infarction is present but lacks specificity and is not often increased without infarction.

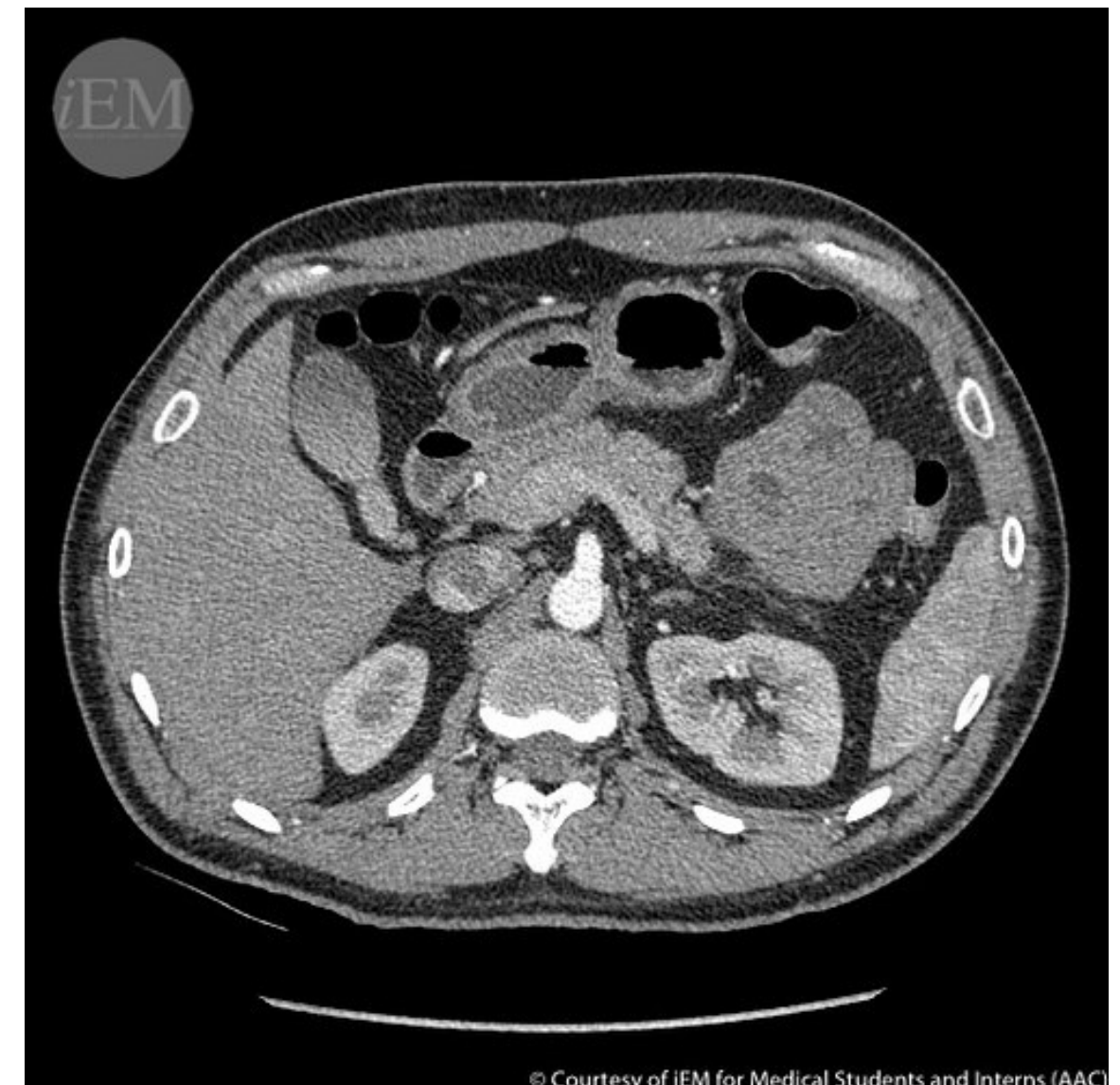
Serum amylase may be moderately elevated in more than half of the cases (lacks specificity).

## Imaging Modalities

Erect Chest x-ray/Abdominal x-ray series used mainly to exclude other causes of abdominal pain or look for complications of acute mesenteric ischemia (e.g., free gas/bowel obstruction). They are often normal in the early stages of acute mesenteric ischemia. However, there are some early and late findings in the X-rays. Early findings are adynamic ileus, distended air-filled bowel loops and bowel wall thickening from submucosal edema or hemorrhage. Pneumatosis of the bowel wall and gas in the portal venous system strongly suggest bowel infarction as late findings.

Multi-detector CT angiography is the primary imaging modality to diagnose acute mesenteric ischemia in the ED. The recent meta-analysis shows a sensitivity of 82.8- 97.6% and specificity of 91.2-98.2 when compared to conventional angiography, which is still considered the gold standard but is rarely available in ED.

**Image 7.8** The CT image shows bowel wall thickness.



The CT angiogram may show edema of the bowel wall and mesentery, abnormal gas patterns, intramural gas, ascites and occasionally direct evidence of mesenteric venous thrombosis. It will also determine other causes for the abdominal pain. If the CT is non-diagnostic and clinical suspicion for acute mesenteric ischemia remains high, there may be a need for angiography or



diagnostic laparotomy depending on institutional practice.

Ultrasound has a limited role in the diagnosis of acute mesenteric ischemia. It is more useful for ruling out other causes of abdominal pain, e.g., cholecystitis, acute abdominal aneurysm rupture, ureteric colic.

## Emergency Treatment Options

### Initial Stabilization and Aggressive Resuscitation

Evaluation of patients with ABC approach gives the physician a chance to recognize immediate life-threatening problems. The most of the patients require supplemental oxygen. But, consider securing airway if needed. Correction of hypovolemia and hypotension (secondary to third space loss and/or bleeding) with normal saline/crystalloids is very important. Because most of the patients have multiple comorbidities, e.g., CHF; there may be a role for invasive hemodynamic monitoring with arterial lines, central venous pressure

monitoring. IVC assessment with bedside Ultrasound can guide the fluid resuscitation before invasive procedures (RUSH protocol).

A quick tutorial on IVC measurement with ultrasound ([video](#))

The ultrasound video shows collapsible and non-collapsible IVC. If the IVC collapse, this means the patient may benefit from fluid resuscitation. ([video](#))

If needed, correct any arrhythmias or CHF which may have contributed to the bowel hypoperfusion. Because one of the predisposing factors is vasoactive agents, discontinue these medications. If pressors are required to support the patient's blood pressure, it is preferable to avoid alpha agonists. In this circumstances, use inotropes at the lowest possible dose. Start broad-spectrum IV antibiotics early, because of the high risk of bacterial translocation across the bowel wall. Patients should not receive anything orally and nasogastric tube placement to

decompress stomach and bowel is often necessary. Correct any electrolyte abnormalities and acidosis.

Urgent surgical consultation should be obtained in the ED as this is a time-sensitive condition. Delays to definitive treatment will result in increased morbidity and mortality. It is best to get a surgical consult when suspicion is high for acute mesenteric ischemia even before a CT angiogram has been done.

### Specific Treatment

In general, the definitive treatment of acute mesenteric ischemia depends on the underlying etiology and the presence or absence of necrotic bowel signs. This ultimately is decided by the surgeons and is one of the reasons why it is extremely important to get an urgent surgical consult when confronted with these patients. In the presence of necrotic bowel/peritonitis, bowel resection will need to be done regardless of which of the four types of the acute mesenteric ischaemic bowel.

In addition, there are some other specific options. Mesenteric artery embolism may benefit from embolectomy then distal bypass graft. Mesenteric artery thrombosis needs bypass graft or stenting. Nonocclusive mesenteric ischemia requires to remove the underlying stimulus and correction of the underlying medical condition. Occasionally direct transcatheter papaverine (vasodilatory) infusion will restore normal blood flow. Mesenteric venous thrombosis showing mild ischemia may be treated with anticoagulation.

## Disposition Decisions

These patients are critically ill with potentially high mortality rates, and as such, they should be admitted and managed in intensive care after surgery.

## The Conclusion of Case

This patient was brought to the resuscitation area of the ED and was put on cardiac and blood pressure monitors and pulse oximetry. After ascertaining that her airway was intact, and providing supplemental oxygen with intranasal oxygen, 2 large bore IV cannulas were established and one liter of normal saline was started, with care taken not to tip her into fluid overload by serial assessment of IVC collapsibility with bedside ultrasound. CBC, renal panel, VBG, and serum lactate, group and crossmatch, and coagulation profile were sent off. Her leukocytes were 12,000 and serum lactate was elevated while the rest of the

results were unremarkable. ECG showed atrial fibrillation (see picture given under the case presentation)

A bedside ultrasound excluded other causes of abdominal pain (e.g., ruptured abdominal aortic aneurysm, acute cholecystitis). Assessment of IVC collapsibility and cardiac ejection fraction gave the clues on aggressive fluid resuscitation. An NG tube was inserted and broad-spectrum IV antibiotics (ceftriaxone and metronidazole) were given. Portable CXR and AXR series were unremarkable. Based on her presenting complaint, a high suspicion for acute mesenteric ischemia (possible acute mesenteric embolism- due to her underlying atrial fibrillation) was entertained and urgent surgical consult was sought. A multidetector CT angiogram showed thickened small bowel wall, dilated bowel loops, and superior mesenteric artery embolism. She was rushed to the operation theatre for exploratory laparotomy as her abdomen was noted to be more tender and had some guarding.

## As a summary, the role of the ED physician is to

- resuscitate the patient as needed,
- make an early diagnosis based on clinical suspicion,
- understand the limitations of laboratory tests in ruling out acute mesenteric ischemia,
- give priority to aggressively resuscitation and
- get urgent surgical involvement

**References and Further Reading**, click [here](#)

# Perforated Viscus

---

by Ozlem Dikme

## Case Presentation

*A previously healthy 42-year-old male presented to the Emergency Department (ED) with a 3-day history of worsening **abdominal pain**. He felt nauseated and vomited twice. His pain started around the umbilicus, moved to the left side of his abdomen and then become generalized. It peaked the last few hours, and the painkillers did not work. His social history revealed that he was non-drinker, non-smoker and did not use any illicit drugs. The past and family histories were unremarkable. His blood pressure was 100/60 mmHg, pulse rate 120/min, the temperature 37.8°C (100°F), and respiration rate 24/min. Physical examination showed diffuse abdominal tenderness and voluntary guarding. Bowel sounds were not heard. Bedside ultrasonography (USG) exhibited increased echogenicity of the peritoneal stripe, with corresponding horizontal reverberation artifacts over the liver. Plain chest*



Audio is available [here](#)

*radiographs confirmed the presence of free abdominal air. Oral intake was stopped, intravenous (IV) catheter was inserted, fluid therapy was started, and cefoperazone sodium was administered intravenously. Blood type and cross, complete blood count and coagulation were ordered. He transferred to the operation theater with the diagnosis of the perforated viscus.*

Can you identify free air on the X-ray?

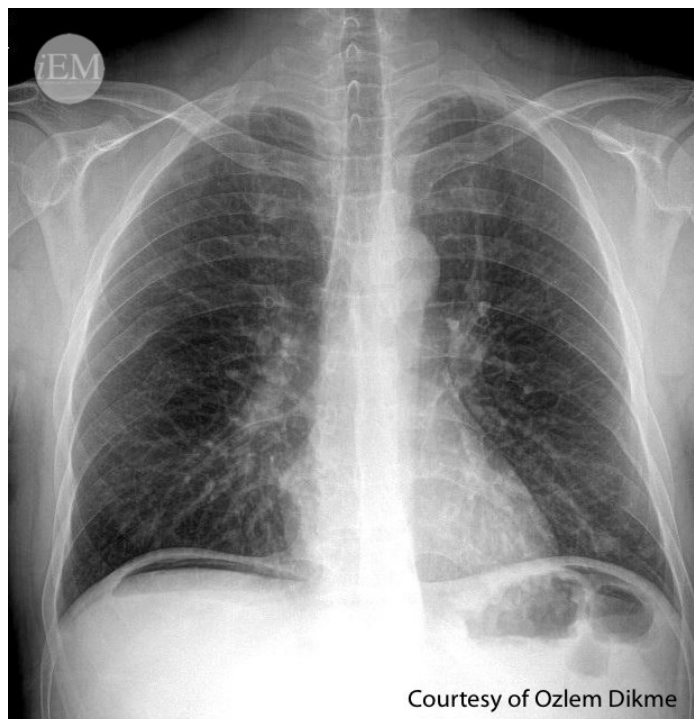
## Critical Bedside Actions and General Approach

All critically ill patients with acute severe abdominal pain is a candidate to have a viscus perforation. The first step is always patient evaluation with ABC approach and stabilization. These patients present to the ED with a severe abdominal pain and discomfort. Because of the pain severity, they may not let you touch their abdomen. They prefer to stand still because of any movement trigger pain. They look sick. Our first priority is to ensure there is no immediate life or organ-threatening situation. If so, immediate actions should be done at the bedside during the initial evaluation. Airway, breathing, and circulation evaluations are completed. However, quick, focused abdominal examination can be done before the full secondary evaluation. Opening two large bore IV lines, fluid therapy, stopping oral intake are some of the routine actions. Patients are attached to the cardiac monitor. Necessary blood samples are collected and sent. The pre-diagnosis of perforated viscus must be explained to the patient, and his approval should be obtained for further evaluation and treatment. The US can be used at the bedside as an adjunct to focused history and physical exam.

## Differential Diagnoses

During the initial evaluation, emergency physicians try to understand possible differential diagnoses in a patient with severe abdominal pain. The below list is given in alphabetical order.

**Image 7.9**



Courtesy of Ozlem Dikme



- Abdominal Aortic Aneurysm
- Acute Cholecystitis or Biliary Colic
- Acute Gastritis or Peptic Ulcer Disease
- Acute MI
- Acute Pancreatitis
- Aortic Dissection
- Appendicitis
- Diabetic Ketoacidosis
- Diverticulitis
- Gastrointestinal carcinoma
- Inflammatory Bowel Disease (Crohn Disease, Ulcerative Colitis)
- Mesenteric ischemia
- Omental torsion
- Rectus sheath hematoma
- Tubo-ovarian pathologies (Ectopic pregnancy, Pelvic inflammatory disease,

Abscess, Endometriosis, Ovarian cyst/torsion, Uterine leiomyomata)

## History and Physical Examination Hints

Thorough medical history usually reveals predisposing factors or possible etiology of perforation. Predisposing chronic conditions include peptic ulcer disease, inflammatory bowel disease, malignancy. Acute conditions like acute appendicitis, acute diverticulitis, infections (e.g., typhoid fever), intestinal ischemia, necrotizing vasculitis and penetrating or blunt injuries may cause perforation. Additionally, caustic substance and foreign body (e.g., toothpicks) ingestions, endoscopic interventions and some medications are associated with perforation. Most common medications are aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs) and steroids.

The patient typically presents with sudden and severe abdominal pain. Asking patients about the characteristics

of pain helps to diagnose. Typical pain starts around a limited location, then expands to all abdomen in a short time. A history of frequent abdominal pain may suggest the patient has a predisposing condition. Free air under the diaphragm may cause referred pain to the either or both shoulders. Vomiting is present in 50% of patients. Shock, sepsis, gastrointestinal or intraabdominal bleeding may accompany perforation.

Ulcer perforation refers to when the ulcer erodes through the wall and leaks air and peptic contents into the peritoneal cavity. The anterior wall of the duodenum is the most common site. Approximately 2 to 10% of patients with peptic ulcer undergo perforation once in their lives.

Contamination of the sterile peritoneal cavity with the chemical and bacterial intestinal content causes inflammation, infection, and sepsis. Therefore, patients may become tachypneic, tachycardic and hypotensive in short time. Immunocompromised or critically ill

patients with other comorbidities pose a greater risk for perforation. Obscure presentations in these patients may delay the diagnosis.

Fever and tachycardia are common. Typically, initial low-grade fever increases over time. Peritoneal findings are almost always present. Diffuse guarding and rebound tenderness are likely. “Boardlike” abdomen is a late sign. Bowel sounds are found decreased in the majority of the cases.

## Emergency Diagnostic Tests and Interpretation

An essential step of the evaluation is imaging and laboratory tests. Erect chest or left lateral decubitus radiographs may reveal pneumoperitoneum. Bedside US may shorten the time to diagnosis and surgical consultation. If these methods do not confirm pneumoperitoneum, the physician should proceed with computerized tomography (CT) or laparotomy options by discussing with surgery. Laboratory tests are not specific

to diagnose but may exclude the other differential diagnoses.

### Bedside Tests

The US is a highly sensitive modality in scanning for peritoneal free air. Recently attention of this technique has been a rise, and it became a popular rapid diagnostic test in EDs. Characteristic US signs of pneumoperitoneum are the ring down artifact and enhancement of the peritoneal stripe over the liver often caused by fluid trapped between gas bubbles. When experienced hands use the US its sensitivity of pneumoperitoneum achieves an almost 93%. The US [video](#).

### Laboratory Tests

Laboratory studies are generally not specific to diagnose. Use of these tests is valuable for the preparation before the surgery and information about the patient basal status. Type and cross, hemoglobin/hematocrit, platelet and coagulation studies are the minimum tests for this purpose. Additionally, blood

gas analysis, lactic acid, liver and renal function tests, lipase/amylase and urinalysis can guide to diagnose and post-surgical care. WBC count usually elevated owing to peritonitis. Amylase may be elevated; liver function test results are variable.

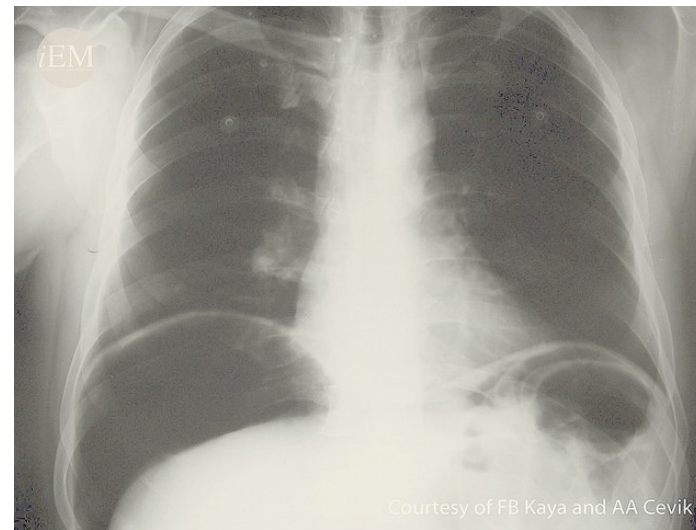
### Imaging Modalities

Plain radiography has a sensitivity demonstrating pneumoperitoneum ranging from 30 to 80%, thus making it is a questionable initial study when a perforated peptic ulcer is considered likely. Free air rises to its highest elevation in the body when the patients sit upright or in positions of left lateral decubitus for at least 10 minutes. Thus it results in increased sensitivity of the radiography. Perforation suggestive findings include subdiaphragmatic free air, visible falciform ligament and air-fluid level. Radiography can be used as an initial screening exam. Thus, a patient may more expediently go to surgery with positive plain radiography. It also has the advantage of being obtainable portably at

the bedside with little interruption in patient monitoring or care. On the other hand, free air cannot be identified in 30% of patients approximately. Thus plain radiography is not sufficiently sensitive to rule out perforation.

Can you identify free air on the X-ray?

**Image 7.10**

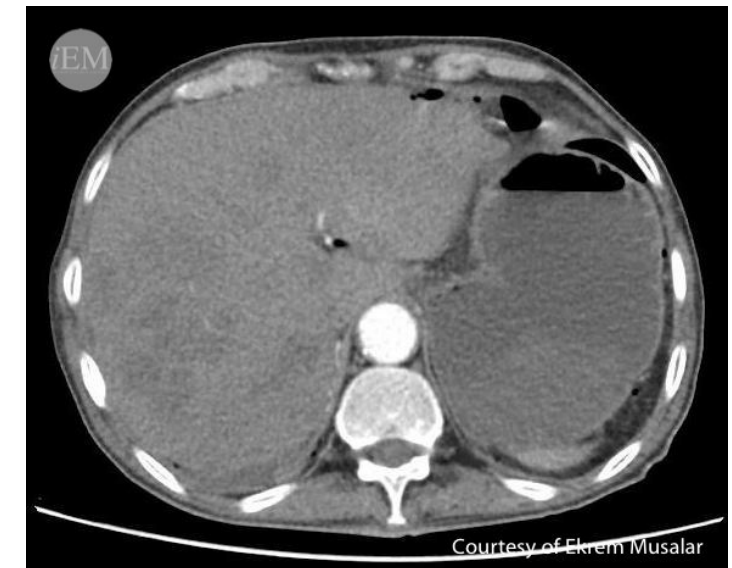


The CT is the most sensitive and specific imaging test in diagnosing a perforated viscus. CT scan has the additional findings of accompanying intra-abdominal abnormalities and etiological changes. It has numerous advantages, first of all, it can detect the small volume of pneumoperitoneum or retroperitoneal free air. Secondly, it can point out that the potential location of the perforation site and known of this may help the surgeon in operation and finally it can provide alternative diagnoses if no pneumoperitoneum is identified. Perforated viscus detection of oral and IV

contrast CT scans has shown as 95 to 98% sensitivity in many protocols.

Can you identify free air on the CT?

**Image 7.11**



## Emergency Treatment Options

The initial management focuses on resuscitation, appropriate antibiotic selection, and immediate surgical consultation. Regardless of the cause, if signs of intestinal perforation with peritonitis are present, prompt emergent laparotomy is indicated. The critically ill patient with a suspected perforated

viscus should be in the ED resuscitation area with two large IV line, oxygen, and close monitoring. Crystalloid fluids and antibiotics are medical treatment essentials. In the emergency setting, antibiotics should cover gram-negative, gram-positive and anaerobic pathogens. Two sample regimens are below. Fore more regimens, and please visit given links under the references and further reading.

**Table 7.7** A Sample Antibiotic Regimen In Perforated Viscus

ANTIBIOTIC REGIMEN	PREGNANCY CATEGORY	DOSAGE
Ceftriaxone and	B	Adult: 1-2 gr IV (bid) Pediatric: 50-75 mg/kg/day IV (bid)
Metronidazole	B	Adult: Loading dose: 15 mg/kg IV (max: 4 grams), 7.5 mg/kg IV (bid or tid) Pediatric: 15-30 mg/kg/day IV (bid or tid) (Check dosage for neonatal of children <2 kg)
Meropenem	B	Adult: 1-2 gr (tid) Pediatric: 20 mg/kg – 1 gr (tid) (Check dosage for children <3 months)

# Pediatric, Geriatric, and Pregnant Patient Considerations

In the pediatric population, two etiologies of perforation are prominent: Blunt trauma and intussusception. Vehicle-related trauma, bicycle handlebar injuries, and seatbelt syndrome are common causes of perforation secondary to blunt trauma in children. Intussusception refers to invagination or “telescoping” of a part of the small intestine into itself. Most cases are children younger than two years. It leads to venous and lymphatic congestion and subsequent intestinal edema. As a result, intestinal ischemia and perforation may occur.

Perforated viscus incidence increases with advancing age. History of peptic ulcer disease or diverticular disease is common in elderly. Medicine-related perforation is common in the geriatric population. NSAIDs increase the risk of colonic perforation in patients with diverticular disease. In an elderly with lower abdominal pain, the physician should suspect perforated diverticulitis or appendicitis.

## Disposition Decisions

All patients require intensive care unit admission. The majority of patients with perforated viscus require laparotomy to explore the whole gastrointestinal system, remove spilled ingredients and repair the lesion. Selected self-closing lesions such as a duodenal perforation covered by omentum may be an exception. They may not need emergent laparotomy but close monitoring and

intravenous large-spectrum antibiotics treatment. Alternative methods of source control such as the use of endoscopic clips for iatrogenic colon injury during colonoscopy are under investigation.

**References and Further Reading**, click [here](#)



## Chapter 8

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# Selected OB&GYN and Genitourinary Emergencies



# Ectopic Pregnancy

---

by Dan O'Brien

## Case Presentation

*A 24-year-old woman presents to the emergency department with the complaint of lower abdominal pain and vaginal spotting. She has never been pregnant. Her last normal menstrual period was two months ago. She had light spotting last month and states that her period this month is late.*

*Her history is notable for one episode of lower abdominal pain two years ago thought to be the pelvic inflammatory disease that responded to a two-week course of oral antibiotics. She has no medical allergies and is not on any medications.*

*Review of systems and family history are unremarkable. Her social history is significant in that she is in a monogamous relationship and is not using birth control.*

*Her general appearance is that of a well-developed female with a temperature of 37°C, a blood pressure of 110/70 mm*



Audio is available [here](#)

*Hg and a pulse of 90 bpm. An examination of her abdomen reveals normal bowel sounds, no masses, distension, organomegaly or rebound tenderness. She is mildly tender to palpation in the left lower quadrant. Pelvic exam reveals left adnexal tenderness without palpable masses. The rectal exam is normal with hemoccult negative stool.*

*Pertinent lab values: urine dip pregnancy testing is positive, quantitative serum B-hCG is 2000 mIU/mL, hemoglobin 13 gr/dL, hematocrit 40%. She is Rh-positive. A transvaginal ultrasound performed by the emergency physician during the pelvic exam fails to demonstrate an intrauterine pregnancy. There is a small amount of fluid in the rectouterine cul-de-sac. 2 cm ectopic pregnancy was identified.*

*Two large-bore IV's were started, the patient was crossmatched for blood and OB-GYN was*

*consulted.  
discussed.*

*Treatment options were*



## Introduction

An ectopic pregnancy occurs when a fertilized egg implants somewhere other than the main cavity of the uterus. The true incidence worldwide is uncertain; however, in the United States, the incidence ranges from 2.7 to 6 deaths per 10,000 live births. Approximately 1%-2% of pregnancies in the United States are ectopic and ectopic pregnancy accounts for 3%-4% of pregnancy-related deaths. Ectopic pregnancy remains the leading cause of maternal death in the first trimester of pregnancy and is the second leading cause of maternal mortality. Early diagnosis and appropriate management may prevent serious adverse outcomes and potentially improve subsequent fertility.

## Critical Bedside Actions and General Approach

Given the consequences of missing an ectopic pregnancy, all women of childbearing years with abdominal or pelvic pain with or without vaginal bleeding must have ectopic pregnancy

excluded. Once the diagnosis is entertained, the first step is to determine whether the patient is hemodynamically stable. Most ectopic pregnancies are stable on presentation. Alternatively, any woman of childbearing years with abdominal or pelvic complaints and unstable vitals should be considered to have a ruptured ectopic pregnancy.

An essential step is to determine if the patient is pregnant. The easiest method is to determine the presence of the  $\beta$  subunit of human chorionic gonadotropin ( $\beta$ -hCG) in the urine or serum. Qualitative urines tests can be easily performed at the bedside. Urine testing is positive when  $\beta$ -hCG is greater than 20 mIU/mL in the urine. Although dilute urine may reduce sensitivity, at this level of detection, the false negative rate will be less than one percent. Quantitative serum testing should be obtained as well since the serum  $\beta$ -hCG level may assist subsequent disposition.

It is important to determine the Rh factor status of the mother. An ectopic pregnancy can sensitize an Rh factor negative mother to Rh-positive fetal blood. Obtaining a type and screen on a stable patient is the most efficient method. A baseline complete blood count is warranted.

Other causes of abdominal or pelvic pain with vaginal bleeding in the first twenty weeks of pregnancy include abortion, implantation bleeding, and gestational trophoblastic disease. Abdominal or pelvic pain causes without bleeding may include gallbladder disease, appendicitis, and hyperemesis. Urinalysis, electrolytes, and liver function studies should be considered.

## Differential Diagnosis

Abdominal or pelvic pain is a common complaint in the emergency department with an extensive differential. All patients who are in childbearing age should be suspected and investigated for appendicitis, endometriosis, ovarian cyst,

ovarian torsion, pelvic inflammatory disease, renal colic, and urinary tract infection. In the pregnant patients, however, intrauterine pregnancy, implantation bleeding, threatened abortion, inevitable abortion, corpus luteal cyst, molar pregnancy, and ectopic pregnancy possibilities should be evaluated.

## History and Physical Exam Hints

*“A 24-year-old woman presents to the emergency department with the complaint of lower abdominal pain and vaginal spotting. She has never been pregnant. Her last normal menstrual period was two months ago. She had light spotting last month and states that her period this month is late.”*

From the emergency physician perspective, the differential diagnosis of a woman with a positive pregnancy test and abdominal or pelvic complaint is broad and will require, in almost all instances in the first trimester, an abdominal and pelvic exam.

Although abdominal pain is reported in 90% of ectopic pregnancies, vaginal bleeding in more than half and menstrual irregularities in up to 70%, none of these symptoms narrow the diagnosis enough to include or exclude the diagnosis of ectopic pregnancy reliably. The classic triad of abdominal pain, vaginal bleeding and amenorrhea is not specific for ectopic pregnancy and, in fact, occurs more frequently with other more common complaints such as spontaneous abortion. Therefore, ectopic pregnancy should be considered in any women of childbearing age who presents with an abdominal or pelvic complaint.

*“An examination of her abdomen reveals normal bowel*

*sounds, no masses, distension, organomegaly or rebound tenderness. She is mildly tender to palpation in the left lower quadrant. Pelvic exam reveals left adnexal tenderness without palpable masses. The rectal exam is normal with hemoccult negative stool.”*

Unfortunately, the physical exam may not be helpful in distinguishing the ectopic pregnancy from other causes of abdominal or pelvic symptoms. In cases of ruptured ectopic pregnancy, a patient may present in shock, with peritoneal signs on the abdominal and pelvic exam. Vital signs are likely normal. The abdominal exam may be nonspecific, the pelvic exam may reveal normal or minor cervical motion tenderness. The uterus may be normal size, and there may be minimal bleeding in the vaginal vault. In



fact, patients with unruptured ectopic pregnancies may present identically as a healthy pregnancy.

## Emergency and Diagnostic Tests and Interpretations

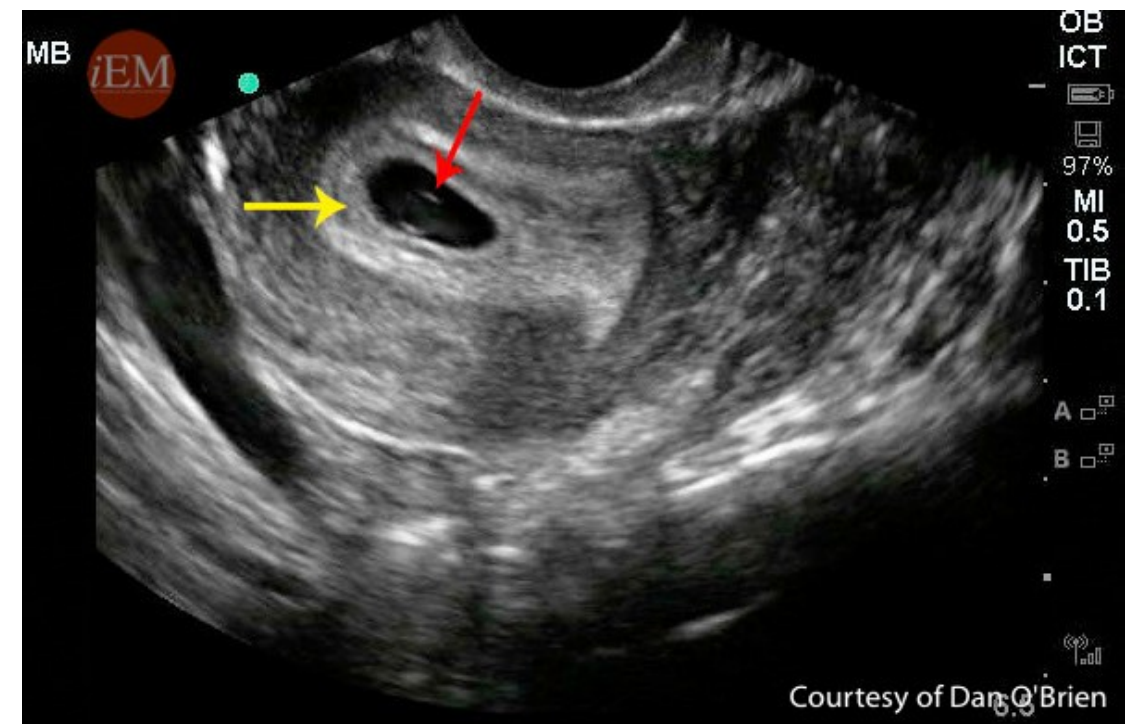
*“Pertinent lab values: urine dip pregnancy testing is positive, quantitative serum B-hCG is 2000 mIU/mL, hemoglobin 13 gr/dL, hematocrit 40%. She is Rh-positive.”*

The patient is pregnant. The primary goal at this point is to determine if an intrauterine pregnancy (IUP) is present. From the emergency physician perspective, an intrauterine pregnancy proven by transabdominal or transvaginal sonography may safely rule out an ectopic pregnancy. She is Rh positive and therefore not at risk for alloimmunization.

Advances in ultrasound imaging and the capability of emergency physicians to perform transabdominal and transvaginal imaging have enhanced patient safety and improved clinical accuracy. There is no definitive guideline regarding sequencing the transabdominal and transvaginal study. In a stable patient, it is reasonable to perform the transvaginal ultrasound exam with the vaginal exam. The exams complement the other. If there is no IUP identified, it is reasonable to search for free fluid in the abdomen.

If the vaginal exam is delayed or the patient is judged low risk, a transabdominal exam to identify an IUP may suffice.

**Image 8.1** Intrauterine pregnancy. Transvaginal image. Normal early pregnancy. Note yolk sac (red) and intrauterine gestational sac (yellow)

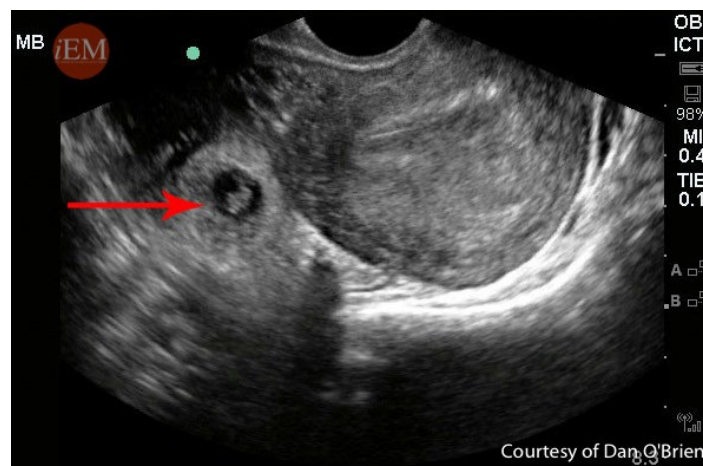


UP had been considered to exclude ectopic pregnancy. The incidence of heterotopic pregnancy may be as low as 1 in 30,000 pregnancies. However, it is as high as 1 in 100 in pregnant who have undergone in vitro fertilization or have taken ovulation-inducing drugs.

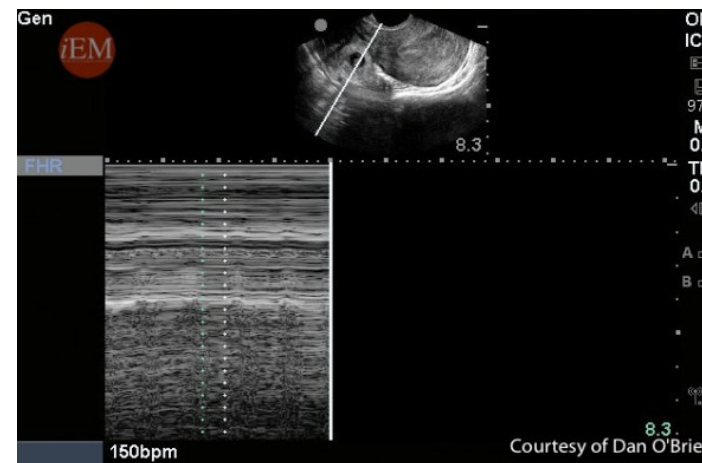
Video shows Ectopic Pregnancy – Transvaginal Ultrasound

*“Transvaginal ultrasound performed by the emergency physician during the pelvic exam fails to demonstrate an intrauterine pregnancy. There is a small amount of fluid in the rectouterine cul-de-sac. 2 cm ectopic pregnancy was identified.”*

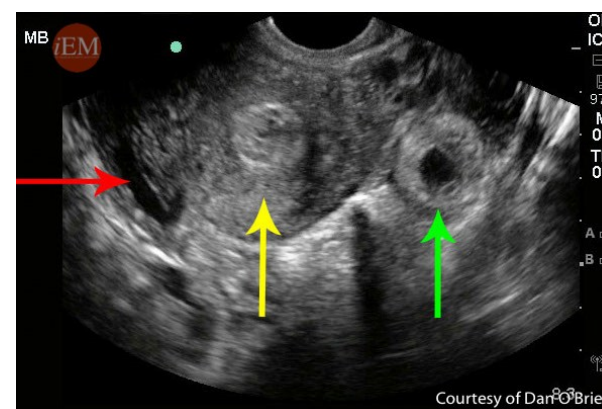
**Image 8.2** Transvaginal ultrasound of embryo in the adnexa next to the empty uterus



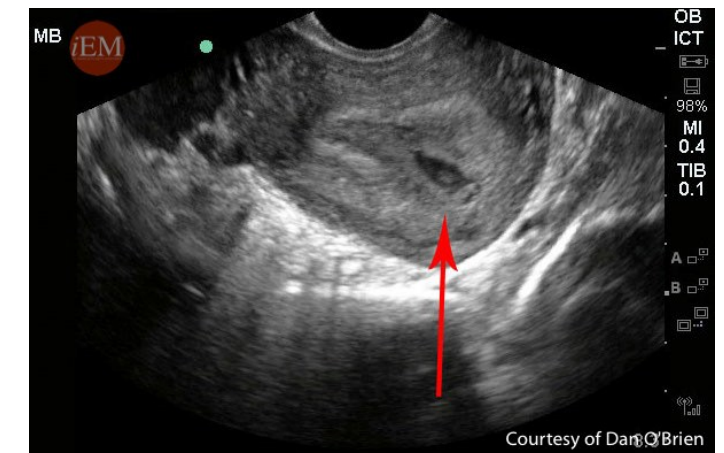
**Image 8.3** Evidence of living embryo on M-mode. M-mode pictorially describes temporal changes at a given depth on one axis while measuring time in the second axis. The fluttering noted is cardiac activity.



**Image 8.4** A small amount of free fluid is noted in the cul-de-sac (red). Don't confuse endometrial reaction typical of pregnancy (yellow) for a gestational sac. An ectopic pregnancy (green) is noted in the adnexa.



**Image 8.5** A pseudo-gestational sac (red), is a collection of intrauterine fluid and may be confused with a true gestational sac. A true gestational sac is normally embedded in the endometrium rather than in the uterine cavity, contains a yolk sac typically seen at 5.5 weeks and has a characteristic double ring or double decidual sign at 4-6.5 weeks.



Laparoscopy should be considered in patients with suspected ectopic pregnancy and nondiagnostic vaginal ultrasound. It is both diagnostic and therapeutic.

Culdocentesis involves extracting fluid from the rectouterine pouch posterior to the vagina through a needle. It has been supplanted by the  $\beta$ -hCG and ultrasound but may be useful when ultrasound is not available.

## Emergency Treatment Options

“Two large-bore IV’s were started, the patient was cross-matched for blood and OB-GYN was consulted. Treatment options were discussed.”

Ectopic pregnancy requires consultation with OB-GYN. If the patient is unstable; resuscitation, urgent consultation, and laparoscopic or open surgery are indicated. In this instance, IV access was established, the patient was typed and cross-matched for blood. The OB-GYN surgeon elected laparoscopic surgery. Ectopic pregnancy was confirmed in the left fallopian tube which was successfully removed.

If the patient is stable and the ectopic is early ( $\beta$ -hCG levels < 3000 mIU/mL) the

consulting OB-GYN physician may consider medical management with methotrexate. The surgeon, not the emergency physician, should decide the treatment.

## The Discriminatory Zone

If the urine  $\beta$ -hCG is positive, but the transvaginal ultrasound does not demonstrate an IUP, the emergency physician should consider a concept known as the “discriminatory zone.” The discriminatory zone is the level of serum  $\beta$ -hCG above which an examiner should be able to see an IUP. With transvaginal ultrasound, an IUP should be seen with a  $\beta$ -hCG level above 1500 mIU/mL and with transabdominal above 6000 mIU/mL. If the serum  $\beta$ -hCG is above 1500 mIU/mL and transvaginal sonography does not identify an IUP, consultation with OB-GYN is essential. These patients should be presumed to have an ectopic pregnancy. Additional diagnostic techniques may include laparoscopy or dilation and curettage.

If the serum  $\beta$ -hCG is below 1500 mIU/mL, the patient is at low risk, and with the concurrence with OB-GYN consultant, the patient may be discharged with follow-up in two days for reexamination and repeat  $\beta$ -hCG levels.

$\beta$ -hCG levels rise rapidly during the first ten weeks of pregnancy then plateau. Although pathologic pregnancies often have lower  $\beta$ -hCG levels than normal pregnancies, there is significant overlap, and absolute levels are not helpful in distinguishing a normal from abnormal pregnancy. A general advisory rule is that  $\beta$ -hCG levels double every 48 hours in a normal pregnancy. However, even here there is significant variation and some controversy. In stable patients, serial measurements and repeated sonography may be used to raise or lower suspicion of an occult ectopic pregnancy.

The  $\beta$ -hCG level representing the discriminatory zone is dependent on the technique and capabilities of the examiner and equipment. The



discriminatory zone should not be used to determine viability or treatment plan associated with an IUP.

Documented ectopic pregnancies have presented with a  $\beta$ -hCG level below test resolution. Therefore do not forgo transvaginal ultrasound investigation in any pregnant patient with a serum  $\beta$ -hCG below 1500 mIU/mL.

## Disposition Decision

If an ectopic is diagnosed in an unstable patient, that patient will require resuscitation, urgent consultation, and surgical intervention.

If an unruptured ectopic is diagnosed in a stable patient, the consulting OB-GYN surgeon may consider surgical or medical intervention based on the characteristics of the ectopic, patient risk factors and stability. Many of the patients managed medically may be discharged with close follow up and strict return instructions. Patients treated with methotrexate who return may represent a unique challenge as the pain associated with methotrexate-induced tubal abortion may not be readily distinguishable from a ruptured ectopic.

A consulting OB-GYN should evaluate a pregnant patient with a  $\beta$ -hCG above the discriminatory zone but without evidence of IUP. Laparoscopic surgery is often diagnostic and, in the case of an ectopic pregnancy, is often therapeutic.

A pregnant patient with a  $\beta$ -hCG below the discriminatory zone and without evidence of IUP may be discharged with the concurrence of the consulting OB-GYN surgeon for close outpatient follow up and serial exams. A portion of these patients will subsequently be diagnosed as an IUP, an ectopic pregnancy, or a threatened, incomplete or completed miscarriage.

**References and Further Reading**, click [here](#)

# Tubo-Ovarian Abscess

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by Matthew Lisankie, Charlotte Derr, Tomislav Jelic

## Case Presentation

*A 19-year-old female presents to the emergency department (ED) complaining of 48 hours of worsening, stabbing left lower quadrant abdominal pain. The patient notes an intermittent, foul-smelling vaginal discharge for the past week. She also endorses fever, nausea, vomiting, dyspareunia, dysuria, and generalized fatigue. The patient is sexually active with one male partner and uses combination OCPs in conjunction with inconsistent utilization of condoms. She denies vaginal bleeding, fevers, jaundice, vomiting, constipation, or diarrhea. Her last menstrual period (LMP) ended 16 days ago and was typical of her usual menses. The patient has a history of menarche at 14 and coitarche at 17. She denies any use of tobacco but admits intermittent alcohol and marijuana use. She has no past medical or relevant family history. There are no known drug allergies.*



Audio is available [here](#)



*Physical exam reveals a well-developed female in mild discomfort but no acute distress. Her vitals are unremarkable except for a temperature of 38.5 and a heart rate of 102. Her abdominal exam reveals moderate tenderness to palpation, worse in the left lower quadrant, with no rebound tenderness. There is no costovertebral angle tenderness, Rovsing sign or McBurney point tenderness. External genitalia is unremarkable. A pelvic exam demonstrates foul purulent discharge in the vaginal vault emanating from the cervical os*

*with no visible blood products. Cervical motion tenderness and pain on palpation of bilateral adnexa are present. Left adnexa is more tender and has a palpable mass on it.*

## Introduction

Tubo-ovarian abscess (TOA) is a walled-off infection of adnexal structures, typically the fallopian tubes or ovary and occasionally adjacent intra-abdominal structures. It is a potentially life-threatening progression of the pelvic inflammatory disease (PID). Thus, TOA and PID share a great deal of pathophysiology and clinical manifestations. TOA is common in women of childbearing age, who have multiple sexual partners and a history of PID [3]. Transvaginal ultrasound is the first choice to diagnose TOA. But, CT remains an important tool in determining further management. [2] Up to 70-80% of appropriately selected TOA cases resolve with appropriate antibiotics alone. However, many patients require either image-guided drainage or surgical exploration for resolution.

## Critical Bedside Actions and General Approach

Assessment of the undifferentiated patient with a high suspicion for tubo-

ovarian abscess begins with the measurement of vital signs and establishment of vascular access.

Continuous cardiac and pulse oximetry monitoring is often prudent, especially if the patient appears distressed or toxic, or has vital signs that fulfill Systemic Inflammatory Response Syndrome (SIRS) criteria.

Rapid determination of the patient's pregnancy status is critical. A positive result warrants immediate rule out of ectopic pregnancy and septic abortion. Additionally, it determines the appropriate interventions and diagnostic modalities. A thorough history and physical including pelvic exam are crucial to timely diagnosis and intervention. If available, bedside transabdominal and endocavitary ultrasound can be a powerful adjunct to the initial assessment of the patient with undifferentiated low abdominal or pelvic pain.

Consider the following critical actions to make a diagnosis and initiate effective treatment:

- Obtain a urine specimen to rule out cystitis and pyelonephritis. It may provide evidence for or against nephrolithiasis. It may determine pregnancy status and therefore, change the choice of radiologic modalities.
- Obtain basic lab work, namely complete blood count (CBC), blood urea nitrogen (BUN) and creatinine. CBC may provide information on the infection and anemia. BUN and creatinine determine if the patient can safely undergo contrasted imaging studies if required.
- Check serum lactate and venous blood gas if there is a concern for sepsis.
- Obtain blood and other indicated cultures if the patient is exhibiting signs of SIRS
- Check electrolytes as hemorrhage, intra-pelvic, and intra-abdominal

catastrophes can often lead to severe metabolic derangements.

- Consider checking hepatic and pancreatic function assays. Abnormal values may suggest other etiologies including biliary obstruction, pancreatitis, Fitz-Hugh-Curtis syndrome, or hepatitis.

Next, prepare for the pelvic examination by obtaining:

- A lighted speculum to inspect the vagina and cervix
- Chlamydia/Gonorrhea PCR swabs
- Wet prep swab
- Lubricant
- Gloves

A chaperone/assistant is recommended for both male and female examiners. Always be sure to discuss the major points of and rationale/risks/benefits/alternatives for the exam with the patient.

The initial pelvic exam is critical as it leads the investigation and provides valuable information to consulting physicians. At the minimum, the emergency physician should note the general appearance of external genitalia, any bleeding, discharge, or odors, the appearance of the cervix and caliber of the os, presence or absence of any cervical motion tenderness, and characteristics of the bilateral adnexa, making note specifically of mass, unilateral tenderness, and description of ovaries if palpable.

## Differential Diagnosis

A chief complaint of acute lower abdominal pain in the female of reproductive age necessitates a rapid rule out of multiple surgical and gynecologic emergencies. The emergency physician should consider ruptured ectopic pregnancy, appendicitis, and TOA in the undifferentiated patient. Likewise, diagnoses including bowel obstruction, ovarian torsion, urinary obstruction should be excluded early as failure to

diagnose these may lead to increases in morbidity and mortality. More common but less immediately-threatening diagnoses include constipation, gastroenteritis, colitis, diverticulitis, ruptured ovarian cyst, uncomplicated pelvic inflammatory disease, nephrolithiasis, urinary tract infection. Finally, consideration of pelvic malignancy, particularly in the post-menopausal patient with suspicion for TOA is recommended.

## History and Physical Exam Hints

Presentation of the patient with TOA can vary from the post-menopausal woman with only vague GI complaints to the teenage patient with septic shock and peritonitis from a ruptured abscess.

The typical presentation of TOA consists of abdominal pain, pelvic mass on examination, fever, and leukocytosis. However, a significant portion of patients with TOA may lack one or more of these features. Therefore, emergency physician

should bear a high index of suspicion in females of reproductive age.

The emergency physician should inquire about the sexual history of the patient. Multiple sexual factors and non-safe sex practices are among the risk factors.

Symptoms related to TOA are abdominal pain, fever, vaginal discharge, nausea, and abnormal vaginal bleeding. Physical examination features related to TOA are mucopurulent discharge, cervical motion tenderness, and uterine or adnexal tenderness.

## Emergency Diagnostic Tests and Interpretation

Ultrasound is the first imaging modality to evaluate the female reproductive system due to low-cost and lack of ionizing radiation. Developing a facility with bedside ultrasound can have a profound impact on the patient's course in the ER. A skilled operator with access to an endocavitary probe can incorporate diagnostic imaging into the initial pelvic exam within the first minutes of

evaluation, and potentially shorten the time to effective antibiotics, definitive imaging, consultant evaluation, and disposition.

The computerized tomography (CT) with oral and IV contrast has improved sensitivity. The other advantages of CT are to show more detailed anatomy and rule in or rule out other differential diagnoses.

## Emergency Treatment Options

Initial management of patients with TOA includes stabilization and timely diagnosis. Assess and frequently reassess airway, breathing, and circulation (ABC). Establish IV access to draw blood, enable intravenous contrast CT and administer medications. Sound medical management is the primary concern of the emergency physician. Medical management primarily includes supportive care (e.g., fluid resuscitation, antiemetics, analgesics.) and broad-spectrum antibiotics.

Pregnancy testing is perhaps the most guiding first step in both diagnosis and treatment. A positive result limits the use of CT, raises the possibility of ruptured ectopic pregnancy, and limits the clinician's armamentarium of antibiotics.

## Medications

The mainstay of the medical therapy is antibiotics. TOA is typically a polymicrobial infectious process and necessitates initial broad coverage for anaerobes, aerobes, gram-positive, and gram-negative bacteria.

A summary of common empiric antibiotic regimens and respective pregnancy categories is as follows:

- Cefotetan (cat B) 2 g IV q12h + Doxycycline (cat D) 100 mg IV/PO q12h
- Cefoxitin (cat B) 2 g IV q6h + Doxycycline (cat D) 100 mg IV/PO q12h
- Clindamycin (cat B) 900 IV q8h + Gentamicin (cat D) 2mg/kg IV (load) then 1.5 mg/kg q8h

• Unasyn (cat B) 3g IV q6h + Doxycycline (cat D) 100 mg IV/PO q12h

- Imipenem-Cilastatin (cat C) 500 mg 16h

## Procedures

Evacuation of the abscess will typically be performed by either an interventional radiologist or gynecologist, depending on abscess characteristics and specific institutional policies.

## Disposition Decisions

Signs of peritonitis, sepsis, or toxic appearance suggest ruptured abscesses. These unstable patients need immediate surgical intervention. Stable patients with a high suspicion or radiographic evidence of TOA warrants ward admission for IV antibiotics and serial evaluation by a surgeon or gynecologist. Discharge from the emergency department and outpatient follow up are not recommended because of the risk of sepsis, peritonitis, and loss of fertility.

**References and Further Reading**, [click here](#)

# Testicular Torsion

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by Sujata Kirtikant Sheth

## Case Presentation

*A 16-year-old male was sleeping when he suddenly started to feel left sided lower abdominal pain. He continued to bear through the pain for another 30 minutes until he started to vomit. At this time he decided to go to the nearest hospital, which is about 15 minutes away. When he reached the hospital his vital signs were as follows: BP: 120/60 mmHg, HR: 120 bpm, RR: 20 bpm, Temp 36.5C, Pain 10/10 and SpO2 was 100% on room air. Physical shows a swollen right scrotum with significant tenderness. What is the next step?*



Audio is available [here](#)



## Critical Bedside Actions and General Approach

- Perform full physical examination including a genital examination
- Induce cremasteric reflex- stroke the inner thigh to see if the cremasteric muscle contracts and the testicle elevates
- Perform ultrasound but do not delay care
- Detorse the testis from medial to lateral
- Consult urology

## History and Physical Exam Hints

Key questions to establish an accurate diagnosis are:

- What time did this occur?
- What was the patient doing at the time?
- Have they had pain on the same side before?
- Was there any trauma involved?

- What are the other associated symptoms?
- Are you sexually active?
- Are all your immunizations up to date?

## Signs and Symptoms

The diagnosis of testicular torsion is based on signs and symptoms. The most classic presentation is a severe testicular pain within 6 hours. However, some patients have milder, less acute pain or no scrotal pain. Alternatively, abdominal pain or inguinal pain may be present. The pain may start at rest, while asleep, or with physical activity. Testicular torsion rarely may be bilateral.

The most common physical findings are testicular tenderness and absence of the cremasteric reflex. Note that 10% of proven testicular torsion cases have a cremasteric reflex. A negative Prehn's test (relief of pain with elevation of the testes) is another sign to look for, but it is not 100% reliable. All patients with testicular torsion have one or more of the following:

Nausea or vomiting, pain duration of less than 24 hours, high position of the testis or abnormal cremasteric reflex.

## Differential Diagnosis

- Epididymal appendage torsion
- Epididymitis or orchitis
- Hydrocele
- Idiopathic scrotal edema
- Idiopathic testicular infarction
- Testicular torsion
- Testis tumor
- Traumatic hematoma
- Urolithiasis

Acute scrotum warrants the exclusion of the testicular torsion. The emergency physician should bear a high index of suspicion because the clinical signs and symptoms vary widely between patients. No single details in the history, physical or imaging confirm or exclude the diagnosis with 100% certainty.

## Pathophysiology

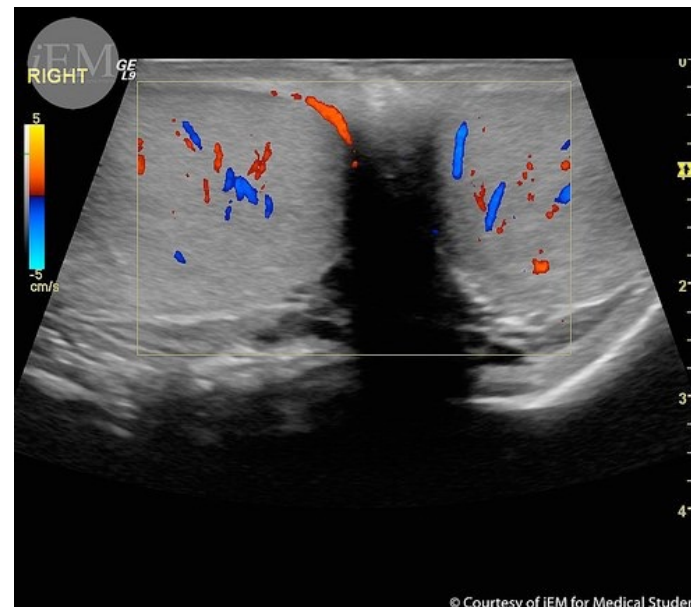
Two main types of testicular torsion are intravaginal and extravaginal. The extravaginal torsion occurs in the perinatal period before the fixation of the tunica vaginalis. Intravaginal torsion refers to when the testis twists inside the tunica vaginalis. Bell Clapper deformity refers to a partial or complete fusion of the tunica vaginalis along the epididymis. It causes an excessive testicular movement and about 12% of the testicular torsions.

## Emergency Diagnostic Tests and Interpretation

Scrotal ultrasound is the method of choice. The ultrasound shows a hypoechoic and enlarged testis in patients with testicular torsion. Reduced blood flow and parenchymal heterogeneity are the other signs of testicular torsion.

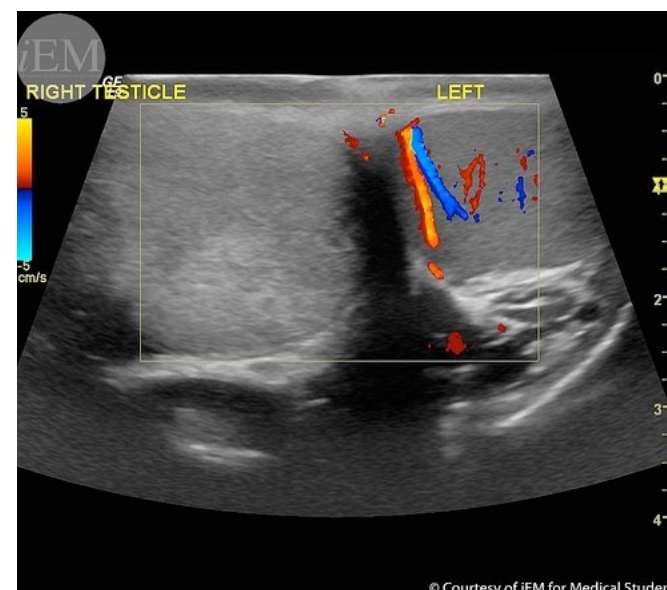
The testicular ultrasound shows bilateral normal blood supply in doppler investigation (below).

Image 8.6



Which testicle has no “blood flow” on the below ultrasound image?

Image 8.7



Urine analysis may prove other diagnoses. However, the presence of infection in urine does not exclude the testicular torsion.

Pre-operation labs if required by the surgical team

## Emergency Treatment Options

### Initial Stabilization

There are a few steps to perform in the ED. The emergency physician may try to detorse the testicle manually at the bedside if the diagnosis is likely. Most testicular torsions are from lateral to medial, so the physician should move the testicle from a medial to a lateral position to detorse the testicle. The patient's pain should relieve if the detorsion is successful. Rarely, if the patient's torsion is from medial to lateral, the physician should detorse in the opposite direction. Turning the testicle in the wrong direction increases the pain.

Torsion of the appendix is more common than torsion of the spermatic cord. Torsion of the appendix is managed conservatively unlike the torsion of the spermatic cord. Torsion of the spermatic cord requires early surgical exploration because this will result in ischemia, damage or loss.

## Medications

Testicular torsion is a painful condition. Please do not ignore and treat the patient's pain with proper pain medication, paracetamol, ibuprofen or with stronger alternatives. In a pediatric patient, the physician may consider options such as intranasal fentanyl. Ideally, oral medications are not preferable as the operation is likely.

## Procedures

If the emergency physician suspects testicular torsion, an emergent urological consult is indicated. The urologist determines the need for ultrasound or emergency surgery. Ultrasound or any other diagnostic tests should not delay

the intervention in patients with high clinical suspicion. The emergency physician may attempt a bedside ultrasound while waiting for the urologist. Consider drawing blood for operation.

## Disposition Decision

### Admission Criteria

Patient with testicular torsion present within 6 hours should undergo an emergent surgery. Patients with testicular torsion for more than 48 hours should be admitted to the urology ward unless the patient is hemodynamically unstable.

### Discharge Criteria

Patients with testicular torsion should not be discharged from the emergency department. In a patient with intermittent symptoms and a negative ultrasound, if the urologist does not admit the patients for observation, it is safer to observe the patient in the ED for repeating symptoms. If the symptoms occur again, repeating the ultrasound and urology consultation is sensible.

## Referral

If you have deemed the patient as not having a testicular torsion you can refer them to urology a week later to see if their symptoms have resolved. Please provide patients with strict information on when to return to the emergency department. If they start having pain again, increased vomiting, inability to urinate, fever, any other worrisome symptoms they need to return.

**References and Further Reading,** click [here](#)



## Chapter 9

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# Selected Neurological Emergencies



# Approach To Patient With Stroke

by Matevž Privšek and Gregor Prosen

## Case Presentation

*A 56-year old female is brought to the ED by the paramedics due to weakness in her left arm and left leg. She is conscious, GCS 15, painless, normal skin color. Vitals are: BP 132/84, pulse 78/min, 14 breaths/min, SpO2 99 %, temperature 36,4 °C, blood glucose 5,4 mmol/L. She says that weakness started about 2 hours ago, while she was watching TV when she suddenly realized she was unable to pick up a glass of juice. She wanted to stand up and almost fell because her right leg did not move. She thought it would go away, but it did not, so she called an ambulance. She denies dizziness, vertigo, nausea, vomiting, headache, visual disturbances. She is otherwise healthy, not taking any medications or drugs. She smokes half a pack of cigarettes daily.*

*The focused neurological exam is performed: pupils are equal and reactive, bulbomotorics and facial mimic seem*



*appropriate except slight drift of right mouth angle. She also has decreased muscle power in her left arm as well as slightly decreased muscle power in her left leg. She denies any sensory deficits. The rest of physical exam is unremarkable.*

*You set up an intravenous cannula, draw some blood for testing, and order emergency non-contrast head CT scan, due to a high suspicion of an acute CVA. The results of the CT scan are back in 35 minutes: radiologist describes no intracranial hemorrhage or ischemic areas. A neurologist is consulted; upon repeated examination, he advises highly for acute ischemic stroke, most likely due to occlusion of the right middle cerebral artery. You immediately start with thrombolysis and transfer her to the neurology ward.*

## Introduction

Stroke or cerebrovascular accident (CVA) is a syndrome of any vascular injury that diminishes cerebral blood flow (CBF) to a specific region of the brain, causing ischemia and thereby consequently causing focal neurologic impairment. Emergency physicians' main goals are early recognition of stroke symptoms, objectification of complaints and prompt diagnostics and treatment.

According to some data, stroke is the third leading cause of death and a leading cause of long-term disability in the United States. Around 2-4 % of hospital admissions are due to potential strokes. Depending on the cause of stroke, in-hospital mortality rates vary between 5-10 % for **ischemic stroke** and up to 45 % for **hemorrhagic stroke**. Up to 50-70% of stroke survivors regain functional independence, while 15-30% be permanently disabled and another 30% eventually require institutional care.

Etiopathogenesis. 80 % of all strokes are ischemic in origin; the rest are hemorrhagic. In the ischemic stroke, a clot stops the blood supply to a specific area of the brain. However, in hemorrhagic stroke, blood leaks into brain tissue. It is highly important to differentiate between them since treatment is completely different.

In the ischemic stroke, a causative clot can originate from large blood vessels of the brain (thrombus) or elsewhere in the body (usually from the heart due to atrial fibrillation; embolus). Rarely,

the cause of ischaemic stroke is hypoperfusion of the brain, due to a systemic problem (e.g., myocardial infarction, dysrhythmias). In hemorrhagic stroke, the main causes are intracerebral (ICH) and subarachnoid hemorrhage (SAH).

Brains are highly sensitive to any alterations in the blood supply of oxygen and glucose that are needed for their metabolism. Immediate alterations in CBF and cellular homeostasis follows a stroke. A complete interruption of CBF (rare) causes loss of consciousness within 10 seconds and death of pyramidal cells follows within minutes. More often, collateral circulation helps to maintain some CBF to the ischaemic region. When CBF drops below a certain point, loss of electrical activity of the affected area occurs, which is clinically seen as a neurologic deficit (but the brain cells remain viable; integrity and function of the neuronal membrane are intact). Area of the brains with electrical silence but viable cells is called penumbra;

irreversible changes have not yet occurred. With further occlusion irreversibility and scope of cerebral infarction increase. Studies have shown that occlusion longer than 6 hours leads to irreversible neurologic deficits.

In hemorrhagic stroke, events beside alterations in CBF, such as red blood cells lysis and increased permeability of the blood-brain barrier lead to brain edema and secondary injury.

## Critical Bedside Actions And General Approach

Regardless of the patients' chief complaint emergency physicians' first task is to rapidly asses patients' condition and vital signs (do not forget blood glucose!), and stabilize them, if necessary. After patients' condition is stable, we continue with establishing chief complaint, focused, but thorough history and physical exam, setting the working diagnosis and list differential diagnoses, and diagnostic and treatment plan. All patients with a suspected CVA

should have their complaints objectified by a focused neurologic exam, and efforts should be made to perform urgent diagnostics.

## Differential Diagnosis

A physician must be well aware of "stroke mimics," which are defined as non-vascular diseases that present with stroke-like symptoms. Since the majority of strokes are treated with thrombolysis, accurate diagnosis due to harmful effects of thrombolytics (significant intracranial bleed in 1 %) is essential.

Possible stroke mimics which may be misdiagnosed as a stroke;

- Migraine
- Seizures
- Psychiatric
- Syncope
- Sepsis
- Brain tumor

- Metabolic
- Transient global amnesia
- Labyrinthitis

Depending on the affected area of the brain and type of stroke CVA can present with a vast list of chief complaints: altered mental status, confusion, syncope, weakness, dizziness, vertigo, ataxia, aphasia, diplopia.

Differential diagnosis of some stroke-related chief complaint

### **AEIOU TIPS: Causes of altered mental status**

A = Alcohol

E = Epilepsy, electrolytes

I = Infection

O = Overdose

U = Urea

T = Trauma

I = Insulin

P = Psychiatric

S = Sepsis, stroke, shock, Syncope

### **HEAD HEART VESSELS: Syncope causes, by system**

#### **CNS causes include HEAD:**

H = Hypoxia/ Hypoglycemia

E = Epilepsy

A = Anxiety

D = Dysfunctional brain stem  
(basivertebral TIA)

#### **Cardiac causes are HEART:**

H = Heart attack

E = Embolism (PE)

A = Aortic obstruction (IHSS, AS or myxoma)

R = Rhythm disturbance

T = ventricular Tachycardia

### **Vascular causes are VESSELS:**

V = Vasovagal

E = Ectopic (reminds one of hypovolemia)

S = Situational

S = Subclavian steal

E = ENT (glossopharyngeal neuralgia)

L = Low systemic vascular resistance  
(Addison's, diabetic vascular neuropathy, calcium channel blockers, anti-hypertensives)

S = Sensitive carotid sinus

### **History And Physical Examination Hints**

The thorough history must be quickly obtained and focus neurological, and general clinical exam must be performed. History should include thorough

“dissection” of the complaint (use modified SOCRATES and SAMPLE), especially the exact time and rate of symptom onset (e.g., sudden onset suggest an embolic or hemorrhagic cause, while gradual onset suggests thrombotic stroke or hypoperfusion). It is also essential to identify any risk factors for thrombotic (hypertension, diabetes, coronary artery disease) or embolic cause (atrial fibrillation, valve replacement, recent MI).

The focused neurological exam can be performed within 4 minutes:

- check mental status,
- cranial nerves,
- motor and sensory function,
- coordination, and
- reflexes

**Table 9.1** Guide For A Quick Neurological Exam

STEP	COMMENTS
mental status	“fogs” family history, orientation, general info, spelling (back & forth), also count backwards from 100 by 3, repeat 7 digit number, recall 3 objects after few minutes
cranial nerves	CN 1: smell tobacco or soap CN 2: visual acuity, gross visual field, ophthalmologic (background) exam CN 3, 4, 6: pupillary light response, lateral and vertical gaze CN 5: double simultaneous stimulation, also corneal reflex CN 8: does he/she hear fingertips moving near ears CN 9, 10: gag reflex CN 11: shoulder elevation CN 12: stick out tongue
motor	drift of upper (and lower, if indicated) extremity hand grasp toe and foot dorsiflexion additional: assessment of individual muscles
sensory	double simultaneous stimulation with needle pin on hands and feet proprioception in big toe additional: check involving dermatomes, light touch, vibration
coordination	finger-to-nose heel-to-shoen rapid alternating movements of hand and feet additional: Romberg, tandem gait
reflexes	biceps (C5-6), triceps (C6-7), knee (L2-4), ankle (S1) Babinski response additional: Kernig and Brudzinski signs

*provided by authors*

In comatose patients, we can perform modified (neurological) exam: vital signs, drop hand overhead, pupils, abnormal eye movements, grimacing, withdrawal from noxious stimuli, Babinski response.

How to clinically differentiate between ischemic and hemorrhagic stroke? Despite clues and suggestions for one cause of symptoms or another, clinical differentiation alone is unreliable! A patient with hemorrhagic stroke typically complains about headache, sudden onset of symptoms that are gradually worsening, nausea and vomiting. Clinical exam often reveals decreased level of consciousness, hypertension, bradycardia, seizures, meningism, fever. Often patients with hemorrhagic stroke present with similar focal deficits as in ischemic stroke, but tend to look sicker. Other clues suggestive of hemorrhagic cause are uncontrolled hypertension, use of anticoagulants, coagulopathies (advanced liver disease), known vascular malformations, brain tumors.

In prehospital setting Cincinnati Prehospital Stroke Scale (CPSS) is highly useful tool to diagnose a potential stroke; if any of tests is abnormal, it suggests possible stroke and this patient should be transferred to hospital as soon as possible. CPSS with 1 abnormal finding has 72 % probability of ischemic stroke and 85 % probability if all 3 tests are abnormal.

**Table 9.2** Cincinnati Prehospital Stroke Scale

FACIAL DROP	ARM DRIFT	SPEECH
person should smile or show his/her teeth  normal: both sides of face move equally	person should close eyes and straight out arms in front for 10 seconds  normal: both arms move equally or not at all	person should repeat a simple sentence  normal: repeats the sentence using the correct word and no slurring

## Emergency Diagnostic Tests And Interpretation

As soon as possibility of stroke has been established, the patient has to be transferred to a facility where emergency non-contrast head CT scan can be performed, mainly to exclude hemorrhage as a cause of symptoms so proper treatment can commence.

Laboratory tests are directed to exclude possible stroke mimics and should include blood glucose, complete blood count, basic metabolic panel, cardiac enzymes, and coagulation studies, as well as EKG and chest X-ray.

American Heart Association recommends that workup should be completed within 3 hours between symptoms onset and beginning of thrombolysis.



**Table 9.3** Recommended Time Frame In Management Of Ischemic Stroke

ACTION	TIME FRAME
Symptom onset to ER doors	< 3 hours
Door to lab work completed	45 minutes
Door to non-contrast head CT ordered	25 minutes
Door to CT being read	45 minutes
Door to decision to give thrombolysis	45 minutes
Door to drug administration	60 minutes (& < 3 hours from onset)

American Heart Association recommendations

An NIH Stroke Scale/Score calculator is a useful tool for quantifying neurologic deficit.

A diagnosis of an acute stroke is often based solely on the patients' history and physical exam, since head CT does not show an acute infarction until at least 6 hours after the occlusion (but it helps to rule out intracranial hemorrhage).

Emergency treatment options. Treatment of stroke is based on the cause of symptoms. If an ischemic stroke is confirmed (or suspected and hemorrhage has been ruled-out) the next step is to determine if a patient is a candidate for thrombolysis.

**Table 9.4** Criteria To Become A Candidate For Thrombolysis

QUESTIONS	YES	NO
<b>all answers must be »yes«</b>		
is the time of onset of symptoms clearly defined?	YES	
will thrombolysis be possible within 4,5 hours of onset?	YES	
has the patient had a good quality of life until now?	YES	
<b>all answers must be »no«</b>		
seizures at the beginning of symptoms?		NO
is this minor/isolated disability (e.g. just dysarthria, ataxia)?		NO
are symptoms rapidly improving?		NO
gastro-intestinal or genito-urinary bleed within the last 3 weeks?		NO
larger surgery within the last 2 weeks?		NO
prior ischemic CVA or severe head injury in the past 3 months?		NO
prior intracranial bleed anytime in the past?		NO
BP > 185/110 (despite therapy)?		NO

## Contraindications for thrombolysis

### Absolute contraindications

- Hemorrhagic (or unknown) CVA anytime
- Ischemic CVA within the past six months
- Malignancy of CNS
- Major (head) trauma or surgery within the past three weeks
- Gastrointestinal bleed in the last month
- Known coagulopathy
- Aortic dissection

### Relative contraindications

- TIA in the past six months
- Peroral anticoagulation therapy
- Pregnancy up to less than a week postpartum
- Refractory hypertension
- Advanced liver disease
- Infective endocarditis
- Active peptic ulcer

- Recent arterial puncture (at the noncompressible site)

If a patient fulfills above criteria for thrombolysis, recombinant t-PA is given at a dose of 0,9 mg/kg IV up to a maximum of 90 mg. 10 % of the dose is given as a bolus, followed by a 60 minutes infusion. Also, blood pressure must be treated before thrombolysis if it exceeds 185/110 (use captopril 12,5 mg SL).

When hemorrhagic stroke is suspected (or confirmed), one must do an urgent consultation with neurosurgeon to decide on further treatment options and plan (e.g., craniotomy and evacuation of hematoma, endovascular aneurysm repair).

## Pediatric, Geriatric, Pregnant Patient, And Other Considerations

Stroke in pediatric population is an extremely rare occasion, but all the principles for adults apply for the pediatric population. Generally, incidence

of stroke increases with age, so physicians have to maintain a high level of suspicion for stroke when managing undifferentiated geriatric patient who is “just unwell.” Stroke in pregnant patient can occur due to predisposition to hypercoagulability, but one must be aware that thrombolysis is contraindicated in pregnancy until the first week after Labor has passed.

## Disposition Decisions

All patients suffering acute stroke should be admitted to stroke care unit or intensive care unit, depending on local policy, abilities and patients’ condition. Patient has to be on a monitor and have frequent assessment of neurologic system.

**References and Further Reading**, click [here](#)

# Acute Ischemic Stroke

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by Fatih Büyükcamlar

## Introduction

Patients with stroke present with sudden onset of paresis, sensory deficits, visual loss or visual field defects, diplopia, dysarthria, facial droop, ataxia, vertigo, aphasia and altered mental status. These symptoms and signs may be seen alone or in combination.

Acute ischemic stroke is a type of brain ischemia due to thrombosis, embolism or systemic hypoperfusion. Atherosclerosis, dissection, fibromuscular dysplasia, arteritis, vasculitis, non-inflammatory vasculopathy, and vasoconstriction may cause thrombosis. The most common source of emboli is cardiac or aortic diseases.

Management principles include stabilization, diagnosing and reversing the cause and decreasing harm to the patient. All patients with acute ischemic stroke have to be evaluated for suitability to thrombolytic therapy or invasive procedures.

# Eligibility criteria for treatment of acute ischemic stroke with recombinant tissue plasminogen activator (alteplase)

## Inclusion Criteria

- Clinical diagnosis of ischemic stroke causing measurable neurologic deficit
- Onset of symptoms <4.5 hours before beginning treatment; if the exact time of stroke onset is not known, it is defined as the last time the patient was known to be normal
- Age  $\geq 18$  years

## Exclusion Criteria

### Historical

- Significant stroke or head trauma in the previous three months
- Previous intracranial hemorrhage
- Intracranial neoplasm, arteriovenous malformation, or aneurysm

- Recent intracranial or intraspinal surgery
- Arterial puncture at a noncompressible site in the previous seven days

### Clinical

- Symptoms suggestive of subarachnoid hemorrhage
- Persistent blood pressure elevation (systolic  $\geq 185$  mmHg or diastolic  $\geq 110$  mmHg)
- Serum glucose <50 mg/dL (<2.8 mmol/L)
- Active internal bleeding
- Acute bleeding diathesis, including but not limited to conditions defined in Hematologic'

### Hematologic

- Platelet count <100,000/mm<sup>3</sup> \*
- Current anticoagulant use with an INR >1.7 or PT >15 seconds\*

•Heparin use within 48 hours and an abnormally elevated aPTT\*

- Current use of a direct thrombin inhibitor or direct factor Xa inhibitor with evidence of anticoagulant effect by laboratory tests such as aPTT, INR, ECT, TT, or appropriate factor Xa activity assays

### Head CT scan

- Evidence of hemorrhage
- Evidence of a multilobar infarction with hypodensity involving >33 percent of the cerebral hemisphere

## Relative Exclusion Criteria

- Only minor and isolated neurologic signs
- Rapidly improving stroke symptoms
- Major surgery or serious trauma in the previous 14 days
- Gastrointestinal or urinary tract bleeding in the previous 21 days

- Myocardial infarction in the previous three months
- Seizure at the onset of stroke with postictal neurologic impairments
- Pregnancy

### **Additional Relative Exclusion Criteria for Treatment from 3 to 4.5 Hours from The Symptom Onset**

- Age >80 years
- Oral anticoagulant use regardless of INR
- Severe stroke (NIHSS score >25)
- Combination of both previous ischemic stroke and diabetes mellitus

(\* Although it is desirable to know the results of these tests, thrombolytic therapy should not be delayed while results are pending unless (1) there is clinical suspicion of a bleeding abnormality or thrombocytopenia, (2) the patient is currently on or has recently received anticoagulants (eg, heparin,

warfarin, a direct thrombin inhibitor, or a direct factor Xa inhibitor), (3) use of anticoagulants is not known. For patients without recent use of oral anticoagulants or heparin, treatment with intravenous tPA can be started before availability of coagulation test results but should be discontinued if the INR, PT, or aPTT exceed the limits stated in above.)

The available data suggest that under some circumstances – with careful consideration and weighting of risk-to-benefit – patients may receive fibrinolytic therapy despite one or more relative contraindications. In particular, there is now consensus that patients who have a persistent neurologic deficit that is potentially disabling, despite improvement of any degree, should be treated with tPA in the absence of other contraindications. Any of the following should be considered disabling deficits:

- Complete hemianopsia:  $\geq 2$  on NIHSS question 3, or

• Severe aphasia:  $\geq 2$  on NIHSS question 9, or

- Visual or sensory extinction:  $\geq 1$  on NIHSS question 11, or
- Any weakness limiting sustained effort against gravity:  $\geq 2$  on NIHSS question 5 or 6, or
- Any deficits that lead to a total NIHSS >5

### **Differential Diagnosis**

Syncope, hypoglycemia, drug toxicity, seizure, intracranial hemorrhagic conditions can be misdiagnosed as acute ischemic stroke.

### **History and Physical Examination Hints**

Most important data of the history is the time onset of symptoms because this is the main data that determine the eligibility for thrombolytic therapy. If the symptom onset time is not known, the time the patient was last awake and free of stroke symptoms is accepted as symptom onset



time. Also, other important data have to be asked like co-morbidities, medications, head trauma and prior stroke.

Stabilization is a priority in every critically ill patient. Once the patient is stable, a focused neurologic examination should be performed. Level of consciousness (LOC), speech, cranial nerve (CN) function, motor and sensory function, and cerebellar function are the main abilities to assess. The physician may assess LOC and speech in a dialogue with the patient. The physician should also check pupillary size, reactivity, and eye movements to assess CN III through CN VI. Additionally, eyebrow elevation and squinting, smiling, gag reflex, shoulder elevation and tongue protrusion are parts of CN evaluation.

As we are still in differential diagnosis process, we have to do full systemic examination including especially cardiopulmonary and neurological examination. Whole body skin should be

controlled for suspicion of trauma. Neurological examination findings give us some clues about the affected or obstructed vascular region.

Next step is to test motor and sensory function. Muscle strength is assessed against resistance. Pronator drift can be tested by having the patient sit with eyes closed and arms outstretched, with palms toward the ceiling, for 10 seconds. Double simultaneous stimulation may be performed by simultaneously touching the right and left limbs. The patient with sensory neglect may feel the right and left sides individually but may ignore one side when both are touched simultaneously.

The last step is to assess cerebellar function, reflexes, and gait. Finger-to-nose and heel-to-shin evaluations, asymmetry of the deep tendon reflexes or unilateral Babinski's sign and observing routine ambulation are all informative parts of the neurologic examination.

## Emergency Tests and Imaging Studies

Hypoglycemia may mimic a stroke. Therefore, fingertip blood glucose measurement is one of the initial essential steps. In case of hypoglycemia (blood glucose level  $<60$  mg/dL), immediate intravenous glucose administration (slow intravenous push of 25 mL of 50% dextrose) is indicated. Whether the patient is hypoglycemic or not, stroke probability should be kept in mind until exclusion.

The basic workup should include an electrocardiogram, complete blood count, plasma urea nitrogen, creatinine, electrolytes, cardiac enzymes, coagulation parameters like prothrombin time, activated partial thromboplastin time and an international normalized ratio (INR). If there is a suspicion for other specific diseases liver function tests, toxicology tests, urinalysis, blood culture,  $\beta$ -HCG, arterial blood gases, lumbar puncture, etc. could be evaluated.

The physician should send the patient to imaging without waiting for the laboratory results.

The evaluation of a suspected stroke patient starts with non-contrast brain computed tomography (CT). A non-contrast brain CT is the fundamental imaging to differentiate hemorrhagic or ischemic stroke. This information determines subsequent treatment.

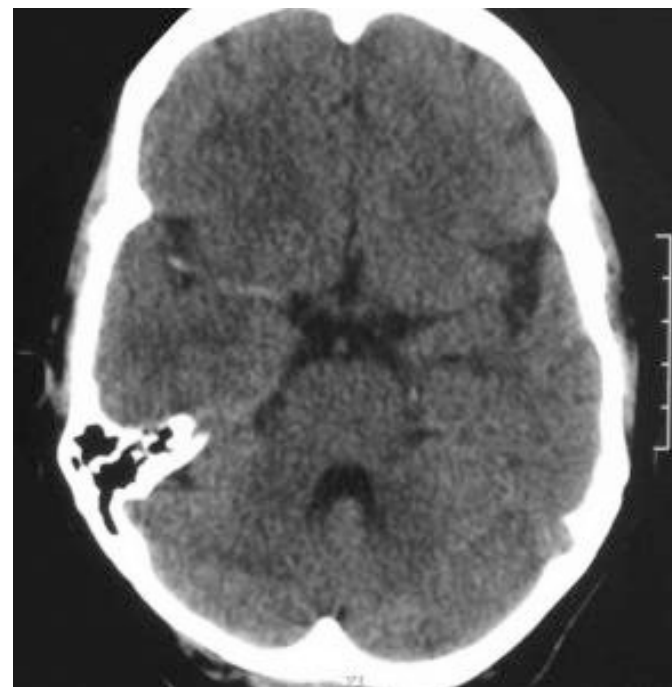
In the acute ischemic stroke, the sensitivity of standard non-contrast CT increases after 24 hours. However, there may be some early signs of infarction in the first six hours.

### Early signs of acute ischemic stroke

- Hypodensity of the lentiform nucleus obscuration
- Hypoattenuation of 1/3 or more of the middle cerebral artery region
- Hyperdensity of large vessels
- Sulcal effacement

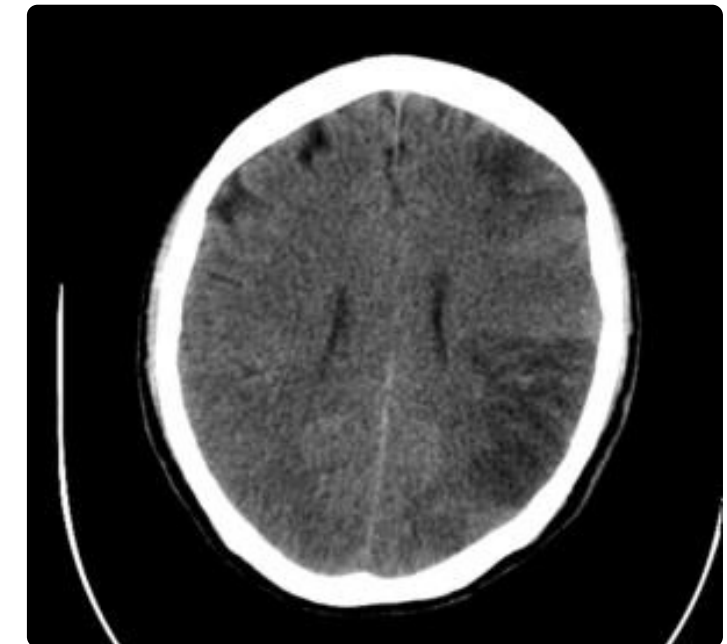
- Hypoattenuation of a focal parenchymal region
- Obscuration of the sylvian fissure and insular ribbon
- The gray-white matter differentiation defect of basal ganglia

**Image 9.1** Hyperdense MCA sign



Case courtesy of Dr Mohammad Taghi Niknejad, Radiopaedia.org. From the case rID: 20784

**Image 9.2** Ischemic stroke



Computed tomographies show two regions of ischemic stroke in the territory of the left middle cerebral artery, involving the regions supplied by both the anterior and posterior branches.

Case courtesy of Dr David Cuete, Radiopaedia.org. From the case rID: 26882

The prevalence of these signs is 61% ( $\pm 21$  standard deviation). The mean sensitivity and specificity of early CT signs are 66% (range 20-97) and 87% (range 56-100) respectively. If the diagnosis is unclear, a diffusion-weighted magnetic resonance imaging (DW-MRI)

may visualize the affected area. DW-MRI can show ischemic changes within 3 to 30 minutes of onset. The apparent diffusion coefficient (ADC) view of DW-MRI gives a quantitative measure of the water diffusion. The decreased water diffusion in cytotoxic edema of acute ischemic stroke causes a hyperintense DW-MRI signal and a hypointense ADC signal. Please see couple examples below.

79-year-old female with left parieto-occipital cortex subcortical white matter infarct. CT imaging of at admission.

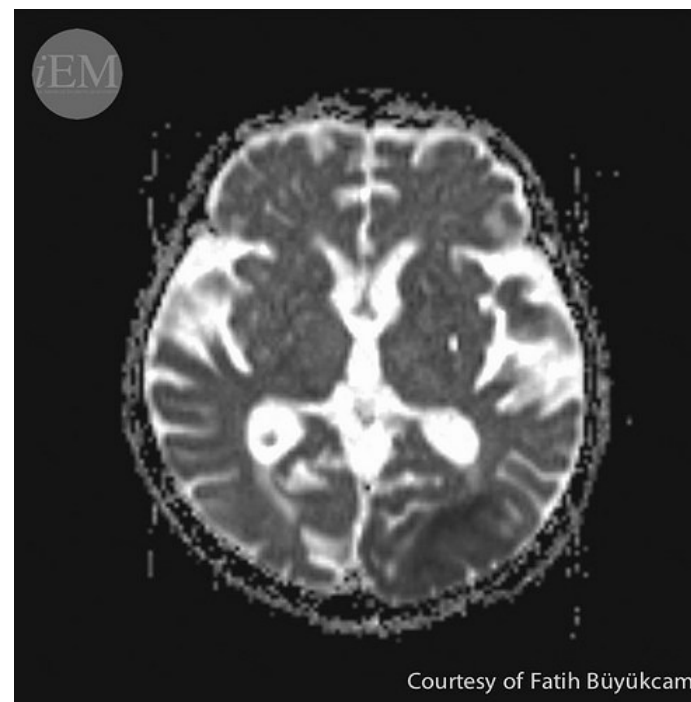
**Image 9.3**



**Image 9.4** CT imaging of at 48th hour



**Image 9.5** DW-MRI imaging of the patient



**Image 9.6** ADC view of the patient



## Emergency Department Management

Oxygen is not recommended for all patients. If the pulse saturation is below 94%, supplemental oxygen should be given.

Fluids should be individualized according to patients' cardiovascular status, plasma glucose, and electrolyte levels. Isotonic fluid is the best choice because hypotonic fluids may aggravate cerebral edema. Both hypoglycemia and hyperglycemia worsen the prognosis;

normoglycemia (60–126 mg/dL) is the best target point. American Diabetes Association suggests achieving glucose targets of 140 to 180 mg/dL in acute ischemic stroke.

Thrombolysis is done in patients eligible for thrombolytic therapy by alteplase, (Figure 10) which is a tissue plasminogen activator. Within 4.5 hours of symptom onset, thrombolytic therapy may be applied.

Apart from brain injury of acute stroke, central nervous system infection, subdural empyema, brain abscess and any concomitant infection may cause fever. Fever is associated with the increased mortality rate, disability, and extended hospital stays. The physician should find and treat the source of fever. Additionally, antipyretics may be used for febrile patients, but its effect on prognosis is still unclear.

#### **Drug of choice for fever:**

Acetaminophen

- <50 kg: 12.5 mg/kg IV q4hr OR 15 mg/kg IV q6hr; not to exceed 750 mg/dose or 3.75 g/day
- ≥50 kg: 650 mg IV q4hr OR 1000 mg IV q6hr; not to exceed 4 g/day Infuse IV over at least 15 minutes

Reducing the blood pressure does not affect the early and long-term outcome. However, systolic blood pressure should be kept under 185 mmHg and diastolic pressure under 110 mmHg to administer thrombolytic treatment. If thrombolytic therapy is contraindicated, the physician should not interfere unless systolic blood pressure >220 mmHg or diastolic blood pressure >120 mmHg or the patient has active coronary artery disease, aortic dissection, hypertensive encephalopathy, acute renal failure, pre-eclampsia, and eclampsia.

The first-line antihypertensive agents are labetalol and nicardipine, and the second-line choice is nitroprusside.

Labetalol

- 20 mg IV over 2 minutes initially, then 40-80 mg IV q10min; total dose not to exceed 300 mg

- Alternative: 1-2 mg/min by continuous IV infusion; total dose of 300 mg has been used

Nicardipine

- Start with 5 mg/hr
- If necessary, increase infusion rate 2.5 mg/hr every 5 minutes to a maximum dose of 15 mg/hr.

Nitroprusside sodium

- Initial: 0.25-0.3 mcg/kg/min IV infusion; may increase by 0.5 mcg/kg/min every few minutes to achieve desired results
- Usual range: 3-4 mcg/kg/min IV infusion, not to exceed 10 mcg/kg/min

Aspirin reduces the 14-day recurrence of ischemic stroke and total mortality. Clopidogrel and aspirin-extended-release dipyridamole can be used alone or with aspirin. Aspirin is the only medication that



is effective for the very early treatment of acute ischemic stroke.

Aspirin (acetylsalicylic acid)

- 160 to 325 mg/day PO
- Aspirin may be given rectally for patients with acute stroke who cannot take by the oral route.
- To the Asian patients with high-risk TIA (i.e., ABCD2 score of  $\geq 4$ ) or minor stroke (NIHSS score  $\leq 3$ ) clopidogrel and aspirin as dual antiplatelet therapy could be given for 21 days 35

Anticoagulation with heparin or low molecular weight heparin is not recommended. It is associated with higher mortality and poor outcomes compared to aspirin alone in 48 hours. However, secondary stroke prevention is recommended for patients with atrial fibrillation and in case of risk for cardiogenic embolism. For this treatment, warfarin may be given in the first 24 hours and continue for two weeks.

## Prognosis

Presence of facial paresis, arm weakness or drift and abnormal speech are the main predictors of outcome. The NIHSS (National Institutes of Health Stroke Scale) score on admission gives a clue about stroke outcome. The use of NIHSS score is recommended for all patients with stroke.

**References and Further Reading**, click [here](#)



# Intracranial Hemorrhage

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by Nur-Ain Nadir and Matthew Smetana

## Case Presentation

*As you start your 3rd night shift in a row, paramedics bring in a 70-year-old female with altered mental status. Patient has a history of hypertension and diabetes mellitus. She is on Coumadin for atrial fibrillation. She was last seen normal three hours ago when she went to sleep. Her husband called emergency medical services (EMS) because she was difficult to arouse. Her blood pressure in the emergency department (ED) is 240/120 mmHg, heart rate 45 bpm, respiratory rate 22 bpm, pulse oxygen saturation 96% and temperature 99°F.1 rectally. On physical examination, she is diaphoretic and unresponsive to any commands. She has dilated fixed pupils. During your assessment, she begins to vomit. What should be your next step in management?*

# Critical Bedside Actions and General Approach

General Assessment: Is the patient Stable or Unstable or in Acute distress?

## Primary Survey

Obtain brief chief complaint and history of present illness from EMS providers or bystanders. Include time of onset and preceding symptoms, i.e., a headache, nausea, vertigo, syncope, chest pain, trauma.

Obtain Vital Signs. Don't forget temperature and blood glucose.

Airway – Is the patient protecting the airway? If not – intubate.

Breathing – Is the patient breathing spontaneously? If not – intubate.

- If the patient is breathing spontaneously but is hypoxic, provide supplemental oxygen.

Circulation – Place two large-bore intravenous lines and check;

- Pulses?
- Blood pressure?
- Skin temperature/quality/moisture/color?

Disability – Check;

- The patient consciousness – Is the patient awake, alert and oriented?
- The patient's score on the Glasgow Coma Scale (GCS)?
- For lateralizing neurological deficits?
- For gaze preference?
- For posturing? – Is decerebrate or decorticate posture present?

Exposure – Completely expose patient.

## Secondary Survey

Perform secondary survey which includes complete examination, history taking following the primary survey and initial stabilization.

Image 9.7 GCS

Glasgow Coma Scale
<b>EYE OPENING</b>
4: Spontaneously
3: To verbal command
2: To pain
1: No response
<b>BEST VERBAL RESPONSE</b>
5: Oriented and converses
4: Disoriented and converses
3: Inappropriate words; cries
2: Incomprehensible sounds
1: No response
<b>BEST MOTOR RESPONSE</b>
6: Obeys command
5: Localizes pain
4: Flexion withdrawal
3: Flexion abnormal (decorticate)
2: Extension (decerebrate)
1: No response

Glasgow Coma Score (GCS) (Modified from Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. The Lancet, 304(7872), 81-84.) - Please read this article to get more insight regarding GCS.

## History and Physical Examination Hints

Obtain a more detailed medical history including surgical, travel, social and medication history. Pay specific attention to risk factors for cerebral hemorrhage.

### Modifiable Risk Factors

- Hypertension- Results in small vessel damage to deeper structures such as basal ganglia and thalamus
- Alcohol- Impairs platelet function and damages endothelial cell wall
- Coagulopathy- Warfarin increases ICH risk up to 2x-4x. Inherent coagulopathic situations such as liver failure increase ICH risk
- Tobacco Use- promotes vascular damage.

### Non-modifiable Risk Factors

- Cerebral Amyloid Angiopathy- Risk increases with age. Amyloid protein deposition weakens vessels' structural integrity

- Age- Directly proportional to ICH risk.
- Structural Abnormalities- aneurysms, connective tissue diseases, congenital arterio-venous malformations (AVMs) and family history of subarachnoid hemorrhage (SAH) increases ICH risk.

## Physical Exam

HEENT exam – Pupils equal round and reactive? Papilledema/Venous pulsations present on fundoscopic examination? Bruising? Raccoon's eyes? Battle sign? Hemotympanum? Ototorhea? Rhinorrhea?

Neck Exam – C Spine tenderness? deformity? Bruising?

Cardiovascular Exam – Bradycardia? Hypertension? Chest Trauma?

Respiratory – Spontaneously breathing? Crackles/Rales? – pattern?

Abdomen – Signs of trauma? Hepatomegaly? Ascites? Signs of cirrhosis?

Skin – Signs of trauma? Needle tracks? medication patches? rashes?

Neurological – complete full neuroexam.

## Differential Diagnoses

- Hemorrhagic stroke
- Ischemic stroke
- Infectious – abscess/meningitis
- Medications – overdose
- Metabolic- HONK, hypoglycemia, electrolyte abnormalities

## Emergency Diagnostic Tests and Interpretation

A non-contrast head CT is typically the first neuro-imaging performed in the ED due to availability, low cost, and high speed. Acute bleeding appears hyperdense in a pre-contrast CT scan. Then the blood starts to appear isodense and hypodense in weeks, respectively. Radiologic signs of elevated intracranial pressure (ICP) include loss of compressible spaces (basal cisterns,

ventricles, cortical sulci), midline shift, herniation, and loss of grey-white matter junction.

Magnetic Resonance Imaging is equally effective for the detection of acute ICH.

However, scanning typically takes longer to perform. It may show the underlying cause of ICH such as a tumor or a vascular aneurysm.

Blood tests should include a comprehensive chemistry panel, complete blood count, coagulation profile, urinalysis. A chest X-ray should be ordered.

## Emergency Treatment Options

### Medications and Procedures

#### Intubation

- Airway protection is the priority. Assess the patient neurologic status briefly before sedation/paralysis. Prefer short-acting sedative and paralytic agents so that frequent neurological examination

can be performed. Protect the patient from hypoxia during the procedure.

#### Impending Herniation

- Clinical signs of elevated ICP may be subtle due to the brain's intrinsic autoregulatory mechanisms or more severe including Cushing's triad (Hypertension, Bradycardia, Irregular respirations), altered mental status, headache, vomiting or focal neurological deficit.
- When elevated ICP is suspected rapid treatment must be performed to decrease the risk of herniation and secondary ischemia.
- Steps to prevent pending herniation:
  - Head elevation to 30 degrees
  - Ensure midline head position to maximize venous drainage.
  - Titrate sedation to reduce agitation
  - Osmotic Agents- Mannitol

- Mannitol (1 g/kg) and hypertonic saline reduce cerebral edema by producing an osmotic gradient that prevents water from moving into the cells during membrane pump failure and drawing tissue water into the vascular space.
- Hyperventilation to produce vasoconstriction.
  - Hyperventilation should be closely monitored. The goal PCO<sub>2</sub> is between 30-35 mmHg. Extreme vasoconstriction secondary to PCO<sub>2</sub> less than 20 mmHg may cause brain ischemia. This should be a temporizing step as injured cerebral blood vessels lose their responsiveness to hyperventilation-induced hypocarbia within 12 hours. Surgical management of the elevated ICP should be the definitive measure.

## Neurosurgical Consultation

- ICH dictates immediate neurosurgical consultation. If hydrocephalus is present, a ventricular drain will allow both measurement of ICP and drainage of cerebrospinal fluid (CSF). Additional hematoma evacuation or decompressive craniotomy may be performed.

## Hypertension

- Blood pressure should be closely monitored. An invasive arterial catheter may be needed. The blood pressure targets in ICH is controversial and depends on the specific type of ICH. Recommended guidelines from the AHA/ASA are illustrated below.
- Recommended Guidelines from the AHA/ASA for Treating HTN in ICH
  1. SBP >200 mm Hg or MAP >150 mm Hg, consider aggressive reduction of BP with a continuous intravenous infusion

2. SBP >180 mm Hg or MAP >130 mm Hg and the possibility of elevated ICP, consider monitoring ICP and reducing BP while maintaining a cerebral perfusion pressure  $\geq 60$  mm Hg.
  3. SBP >180 mm Hg or MAP >130 mm Hg and no evidence of elevated ICP, then consider reduction of BP (e.g., a MAP of 110 mm Hg or target BP of 160/90 mm Hg).
- While the decrease in blood pressure may reduce the hematoma formation and risk of re-bleeding, it may also reduce cerebral perfusion. One should avoid over-lowering blood pressure as significant drops may minimize perfusion to the ischemic penumbra. Rapidly titratable antihypertensive drugs are recommended. Hypotension may be managed by crystalloid fluid, blood or vasopressors to maintain a systolic blood pressure of >90 mmHg.

## Seizures

- Patients with ICH are at an increased risk of developing seizures. This risk increases with lobar hematoma location, hemorrhage size, depressed mental status, history of epilepsy, history of cirrhosis and penetrating trauma.
- The current guideline recommends against the routine administration of antiepileptic medication. However, in case of seizure, antiepileptics such as fosphenytoin (20mg/kg loading dose) should be initiated.

## Anti-coagulation

- Coagulopathy should be reversed. Common anti-coagulants and their reversal agents are illustrated in Table.



**Table 9.5** Common anti-coagulants and their reversal agents

DRUG	MECHANISM OF ACTION	REVERSAL MEDICATION
Warfarin	Inhibits Vitamin K clotting factors (2,7,9,10)	Vitamin K FFP Prothrombin Complex Concentrates
Unfractionated Heparin	Binds to antithrombin 3	Protamine
Low Molecular Weight Heparin	Inhibits factor Xa	Protamine incompletely reverses factor Xa inhibition
Aspirin	Irreversibly blocks cyclooxygenase	Platelet transfusion to increase normal platelet count by 50,000
Clopidogrel	Inhibits ADP receptor on platelet membrane	Platelet transfusion
Dabigatran	Direct thrombin inhibitor	No specific reversal
Rivaroxaban, apixaban	Inhibit factor Xa	No specific reversal

*provided by authors*

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

- Geriatric patients: The elderly are particularly at risk for spontaneous and traumatic ICH due to higher hypertension and brain atrophy prevalence, and frequent use of anticoagulation.
- Pregnant Patients: During pregnancy, ICH risk increases in case of preeclampsia, eclampsia and gestational hypertension.
- Pediatric Patients: The majority of childhood ICH is secondary to trauma. The physician must always look for the signs of non-accidental injury in pediatric patients with ICH even if the history suggests otherwise.

## Disposition Decisions

Patients are typically admitted to an ICU.

**References and Further Reading**, click [here](#)

# Seizure

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by Feryde Caliskan Tur

## Case Presentation

*A 20-year-old female patient presented to the ED with shoulder pain beginning in the morning at work. She had no history of trauma. However, her right shoulder had a deformity. Her X-ray showed a posterior shoulder dislocation, and she could not explain how it occurred. While the emergency physician was making preparations to reduce the shoulder, the nurse shouted that the patient was having a generalized tonic-clonic convulsion.*

## Introduction

The seizure is a frequent neurologic emergency in the emergency department (ED), accounting for 1-2% of all emergency department visits. The highest incidence of seizure is among infants and individuals aged > 75 years. The infantile seizure occurs due to the high prevalence of febrile seizures, and in the elderly, it is mostly secondary to structural brain damage. Most seizures (49%) are related to alcohol or drugs, head injury, and pre-existing diagnosis of epilepsy. Less frequent etiologic pathologies are brain tumors (3%), metabolic abnormalities (3%), stroke (3%), and neurocysticercosis (1%). The reason for the rest (41%) is unknown. Managing patients with seizure and no apparent etiology may be challenging for the emergency physician.

## Critical Bedside Actions and General Approach

The patient was placed immediately in the left lateral recovery position to avoid aspiration of vomitus. Simultaneously, 4

L/hour oxygenation was started via an air mask, and the vascular access was established. Lorazepam 2mg, IV was given by slow injection to stop the seizure.

The seizure stopped in a few minutes. The patient's blood glucose was measured at 122 mg/dL. Her vital signs were: blood pressure 116/80 mmHg, heart rate 60 beats per minute, respiratory rate 12 breaths per minute, oral temperature 98.6 °F, and a pulse oximetry 100 % on room air.

Her physical examination was normal except for the right shoulder. When the patient regained consciousness, she wanted to know what had happened. It was her first witnessed seizure. There was no drug or substance abuse, and her menstrual status was normal. She had frequent headaches for the last month, and she had been evaluated by a neurologist.

## History and Physical Examination Hints

History of seizures, head injury, recent fever (suggests infection or drug reaction), anticholinergic and sympathomimetic syndromes (mainly depending on street drug-abuse) are essential clues of the etiology of seizure.

A full neurological examination should be made. Motor movements and the accompanying eye movements during the seizure may distinguish the seizure from psychogenic seizures (pseudoseizures or nonepileptic seizures). 12% to 18% of patients with transient loss of consciousness are described as psychogenic seizures. It can exist concomitantly in patients with neurogenic seizures. Psychogenic seizures are a manifestation of psychological distress.

Neurologic deficits may be secondary to an old lesion, new intracranial pathology, or postictal neurologic compromise (called Todd paralysis, the physician may rule out a new structural lesion).

Seizure is defined as a sudden change in behavior, characterized by an alteration in sensory perception or motor activity, resulting from an abnormal, excessive, and synchronous electrical firing in groups of neurons, caused by disequilibrium of the neuronal cell membrane, normally kept stable by inhibitory mediators such as gamma-aminobutyric acid (GABA).

Convulsions are the motor manifestations of this abnormal electrical activity. The clinical manifestations of seizures include focal or generalized motor activity, altered mental status, sensory or psychic experiences, and autonomic disturbances.

Epilepsy is referred to convulsive seizures without any recurring or provocative reason.

The postictal period is a change in consciousness that starts before the seizure and lasts for a while.

A generalized seizure is related to both hemispheres accompanied by convulsions in the entire body took place.

Focal seizures took place in certain parts of the body remain localized in a single hemisphere of the brain so may be easily overlooked. A simple focal seizure may or may not cause a depressed mental status, but a complex focal seizure causes changes in consciousness.

Generalized status epilepticus is seizures that prevent the return to conscious state with frequent recurrences or last more than 20 minutes. According to these definitions, our case had a generalized seizure. This definition of seizure can change the patient management.

Pregnant patients with seizure: Pregnant patients of more than 20 weeks' gestation (and up to 6 weeks postpartum) with eclampsia is the major consideration in presentation with new-onset seizures. Gestational epilepsy is diagnosed in approximately 25% of patients with new-onset seizures during pregnancy.

## Emergency Tests and Interpretation

Adult patients with new-onset seizures who are otherwise healthy and have returned to baseline require only simple tests including serum glucose, sodium level, and pregnancy test. In patients with fever, comorbid disease, or new neurological deficit further testing is indicated.

### Bedside test

- Capillary glucose level (stick glucose)
- Blood gases: It may show an anion gap metabolic acidosis secondary to lactic acidosis. Lactate elevates within 60 seconds of a convulsive seizure and normalizes within 1 hour.
- Electrocardiography (ECG) should be obtained in patients with new-onset seizure, or with the suspicion of decreased CNS perfusion secondary to a cardiac cause. In addition to ischemia, conduction abnormalities and dysrhythmias are important disorders to be excluded, see below.

- Differential diagnosis by ECG

### • **Conduction Disorders That Can Be Cause of Seizure-like Activity**

(Adapted from: *Seizure: Emergency Medicine, Second Edition*. Editor; Adams, James G., MD, 2013, 2008 by Saunders, an imprint of Elsevier Inc. Book chapter 99)

- A seizure may also result in dysrhythmia-related syncope.
- Brugada syndrome: Right bundle branch block with ST-segment elevation in leads V1-V3
- Long QTc interval
- Short QTc interval
- Sodium channel blockade with cyclic antidepressants, lidocaine, anticholinergics
- Torsades de pointes
- Widening of QRS complex

- Wolff-Parkinson-White syndrome

### **Laboratory tests**

- CBC (reveal anemia or infectious process)
- Electrolytes (especially Na, and Ca/Mg),
- Serum glucose
- Urea-nitrogen, creatinine,
- Pregnancy tests in women of childbearing age (rule out eclamptic seizures),
- Antiepileptic drug levels,
- Liver function tests, and
- Drugs-of-abuse screening
- Spinal tap is useful to evaluate suspected CNS infection (patients with fever, severe headache, or persistent altered mental status) or HIV/AIDS population (strong suspicion of immunodeficiency).

### **Imaging modalities**

Brain CT is indicated in all first-time seizures. Additionally, new focal neurological deficit, history of trauma, toxic drug and substance use necessitates a brain CT, see below.

### **Indications for CT Scanning of the Brain**

*Adapted from: Teran F, Harper-Kirksey K, Jagoda A. Clinical decision making in seizures and status epilepticus. Emerg Med Pract. 2015 Jan;17(1):1-24.*

- A Persistent change in mental status
- Advanced age
- History/clinical evidence of trauma
- Human immunodeficiency virus/acquired
- Immunodeficiency syndrome (HIV/AIDS)
- Infection (neurocysticercosis)
- New focal neurological deficit
- Suspicion of parasitic central nervous system



First-onset seizures or seizures with persistent mental status change, focal neurologic deficit, or suspicion of organic intracerebral lesion necessitates brain computed tomography (CT). It will help to diagnose epidural or subdural hemorrhage, a brain mass or infections.

### Factors Associated With Abnormal Computed Tomography Findings

(Adapted from: Teran F, Harper-Kirksey K, Jagoda A. Clinical decision making in seizures and status epilepticus. Emerg Med Pract. 2015, page 9)

- Altered mental status
- Closed head injury
- Focal abnormality on neurological examination
- History of cysticercosis
- Malignancy
- Neurocutaneous disorder
- Patient aged > 65 y
- Seizure duration > 15 min

- The absence of a history of alcohol abuse
- The focal onset of the seizure

MRI can reveal additional diagnosis like brain abscess and central vascular events. MRI is more sensitive than CT and can successfully diagnose temporal sclerosis, cortical dysplasia, vascular malformations (e.g., AV aneurysms), and some tumors in addition. Its use is limited in emergency setting.

Electroencephalography (EEG) is important to monitor intubated patients or patients with persistent altered mental status. (Suspicion of nonconvulsive status epilepticus). EEG records brain electrical activity and is used for definitive diagnosis. The need for EEG in the emergent setting is limited and must be saved for when seizure activity is uncontrollable or difficult to diagnose. (e.g., patients who are under sedation or are intubated).

## Emergency Treatment Options

Maintenance of adequate cerebral perfusion, oxygen and glucose supply to the brain, is the goal of initial treatment. Airway must be preserved. Continuous pulse oximetry and capnography should be monitored. Jaw thrust and nasopharyngeal airway ensure an improved oxygenation. Preventing aspiration in the postictal phase, seizure control (administration of anticonvulsants), correction of hypoglycemia, IV line placement, and administration of oxygen can be addressed together with coordinated team care. If there is trauma signs secondary to the seizure, cervical spine precautions (immobilization with a collar) should be initiated.

### Medications

Traditionally, pharmacologic therapy of seizure has been divided into three steps (Table). Generally, benzodiazepines are the initial choice, followed by phenytoin or valproic acid. Levetiracetam is the

second-step choice in patients with liver disease. Third step interventions are infusions of benzodiazepines (midazolam or long-acting lorazepam), propofol, or barbiturates (e.g., pentobarbital; pay attention to hypotension). In up to 30% of the patients, the first- and second-step therapies fail.

- The drug choice is same for nonconvulsive seizures.
- Secondary causes for seizure must be considered for the treatable etiologies (e.g., intracranial infections and lesions, metabolic abnormalities, drug toxicities, and eclampsia).

**Many considerations on the use of medications should be remembered;**

- Use of benzodiazepine for active seizure in the prehospital setting is strongly supported.
- IM midazolam is the best option for the prehospital treatment of seizure, especially when no intravenous access is immediately available.

- If the patient needs intubation, pretreating with lidocaine (1.5 mg/kg) and a low dose (defasciculating dose) of a nondepolarizing paralytic agent (e.g., vecuronium, 0.01 mg/kg) is preferable to control intracranial pressure from trauma or intracranial bleeding. Short-acting paralytic agents such as succinylcholine is recommended during rapid sequence intubation.
- Remember that phenytoin (effective dose 20 mg/kg) must be infused with saline solution (not dextrose due to precipitation). Its main adverse effect is arrhythmia due to QT prolongation. Therefore, the patient must be monitored during the infusion.
- Alcoholic seizure and seizure secondary to isoniazid in tuberculosis treatment are treated with 5 gr IV vitamin B6 in adults and 70 mg/kg IV pyridoxine infusion in children.
- Seizures due to ecstasy or cocaine abuse are treated with benzodiazepines

and aggressive cooling. Phenytoin is not effective in substance-related seizure, and also may be harmful to drug intoxications such as tricyclic antidepressants and antiarrhythmics.

- The clinician should be aware that administration of phenytoin and phenobarbital is rate-dependent and that patients may continue to seize for 30 minutes before effective serum levels are reached.
- Timely administration of antibiotics is important for the survival of patients with infectious problems
- Prophylactic medication is not indicated to prevent late posttraumatic seizures.
- New generation drugs such as lamotrigine (FDA category C) is used for partial, generalized, and absence seizures for maintenance therapy.

**Table 9.6** Antiepileptic drugs and doses for seizures therapies

MEDICATION	LOADING DOSE IV	MAINTENANCE DOSE	PEDIATRIC DOSE	COMMENTS
Diazepam	10 mg over 2 min, or 10-20 mg	Repeat q 5-10 min	0.15 mg/kg IV; 0.2-0.5 mg/kg	respiratory depression, hypotension
Lorazepam	2-4 mg IV	Repeat once in 10-15 min	<13 kg: 0.1 mg/kg IV (max 2 mg); 13-39 kg: 2 mg IV; >39 kg: 4 mg IV	respiratory depression, hypotension
Midazolam	0.1-0.2 mg/kg (also IM, IN rectal or buccal)	0.001 mg/kg/min	0.2 mg/kg IV, IN (max 5 mg); 0.5 mg/kg buccal (max 5 mg); <13 kg: 0.2 mg/kg IM (max 5 mg); 13-39 kg: 5 mg IM; >39 kg: 10 mg IM	respiratory depression, hypotension
Phenytoin	18-20 mg/kg, max rate of 50 mg/min	100 mg IV/PO q 6-8 h, 20 mg/kg IV infusion	20-mg/kg IV infusion	hypotension, ataxia
Pentobarbital	5-20 mg/kg, 25 mg/min	1-3 mg/kg/		respiratory depression
Phenobarbital	10-15 mg/kg bolus, 60 mg/min	120-240 mg q 20 min		respiratory depression, hypotension
Propofol	1-2 mg/kg IV over 5 min	2-4 mg/kg/h		respiratory depression, acidosis (in children)
Valproic acid	20 mg/kg at 20 mg/min	Repeat if needed		subtherapeutic dosages
Magnesium sulfate	4-6 g over 15 min	2 g/h		respiratory depression, loss of deep tendon reflexes
Calcium gluconate, calcium chloride (has three times more Ca <sup>2+</sup> )	10 mL of 10% calcium gluconate in 50-100 mL of D5W, over 5-10 min			Only indicated for hypocalcemia or hyperkalemia
3% NaCl (hypertonic saline solution)	300-500 mL of 3% NaCl in 20 min	Repeat if needed		Only for hyponatremia
Pyridoxine	5 g (50 ampoules of 100 mg of vitamin B6)			Only for some drug-induced seizure

## Pregnant patients with seizure

Seizure related hypoxia and acidosis have a greater teratogenicity potential than anticonvulsant medications. Therefore, actively-seizing pregnant patients may be managed the same as nonpregnant. Magnesium sulfate is the therapy of choice in the treatment of acute eclamptic seizures and for prevention of recurrent eclamptic seizures. Additionally, a seizing chronic epileptic pregnant can be treated with midazolam. Remember that midazolam (FDA category D) is the safest; valproate and phenytoin are the most harmful (both FDA category D) antiepileptic drugs in first-trimester pregnancy.

## Emergency Procedures

Airway management is the most important procedures during a seizure activity. The majority of the patients, however, will not need definitive airway protection. The basic airway maneuvers or adjunct devices may overcome the temporary airway obstruction risks. Simple seizures are self-limiting in most cases. Maintaining the airway by jaw thrust/chin lift maneuvers, inserting an oropharyngeal airway, and oxygen mask ventilation are first measures to prevent the tongue bite, airway obstruction, and apnea. A corkscrew is useful to open the jaw. In cases that oxygen inhalation and intubation fail, a surgical airway is indicated.

## Disposition

## Decisions

### Admission criteria

Patients with persistent seizures, change of mental or neurologic status, or underlying medical conditions that require hospital treatment (e.g., sepsis, overdose, brain trauma) should be admitted. Patients with status epilepticus should be admitted to the intensive care unit. Patients with subtherapeutic drug levels should receive an additional dose before discharge. First-onset seizures should have follow-up arranged with the neurology service/consultant for further investigations. A second attack occurred in 1 month in 32% of patients with a first-onset seizure. Risk factors such as alcoholism, comorbidities or known cardiovascular disease, age > 60 years, history of cancer, or history of immune-compromise should be considered for admission.

### Discharge criteria

Discharge decision should be based on final underlying diagnosis. Chronic seizures can be discharged after return to the baseline neurologic levels.

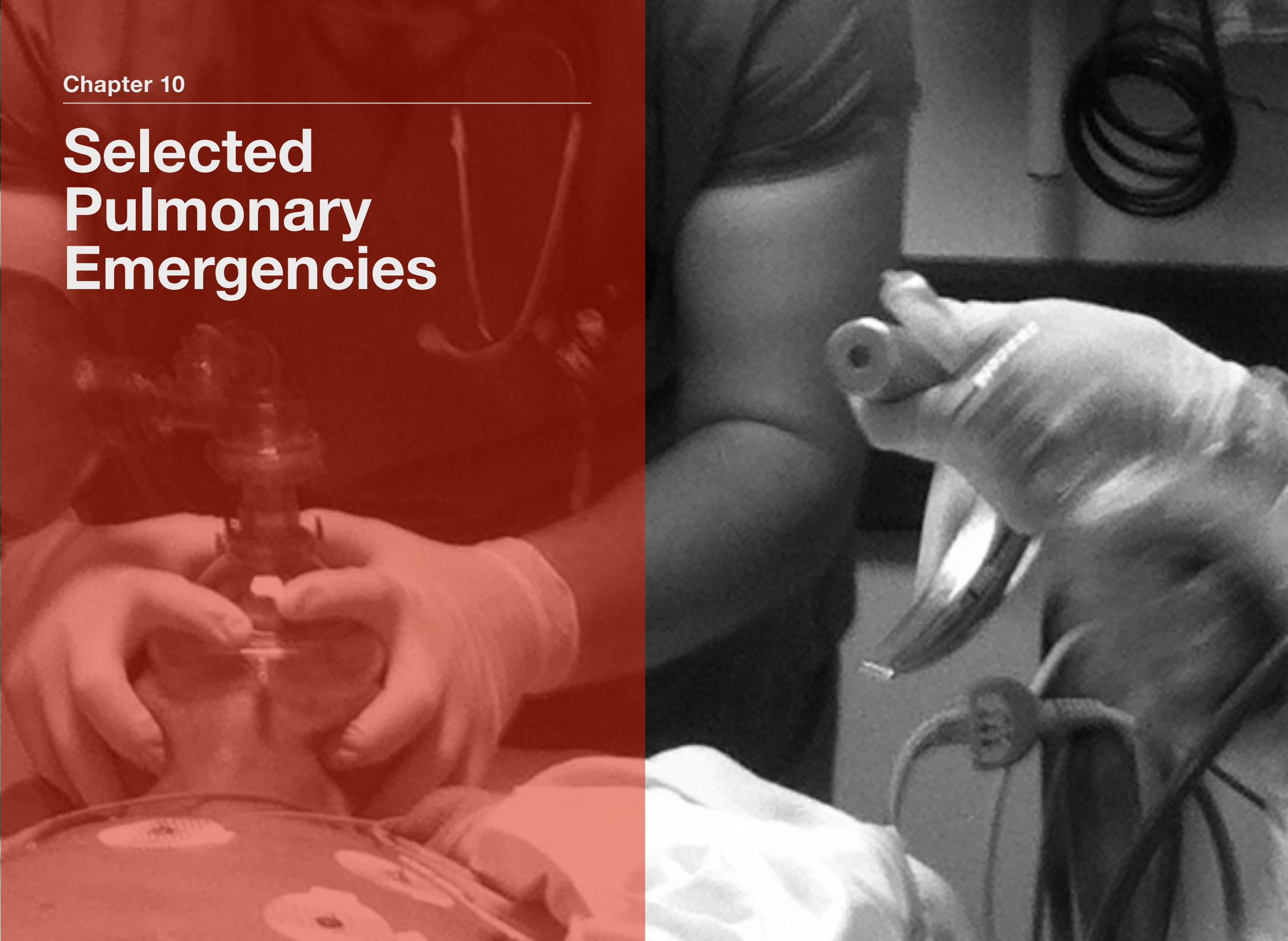
**References and Further Reading**, click [here](#)



Chapter 10

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# Selected Pulmonary Emergencies





# Asthma

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by Ayse Ece Akceylan

## Case Presentation

*A 50-year-old male with a history of asthma presents to the emergency department (ED) with shortness of breath, tachypnea, and audible wheezing. The patient has taken his prescribed medications at home, but his symptoms did not relieve. His vitals were as follows: BP 130/90 mmHg, HR 120 bpm, RR 40 bpm, SpO2 92% on room air. Physical exam revealed accessory respiratory muscle use, expiratory wheezing and decreased breath sounds with expiratory rhonchi. Nebulized short-acting beta2-agonists (SABA) and systemic corticosteroid were ordered. Peak expiratory flow (PEF) measurements before and after treatment were 125 and 360, respectively. Auscultation after initial treatment revealed much-improved airflow. The patient was discharged following clinical improvement, with a prescription of oral corticosteroids in addition to his current medications.*

## Introduction

Asthma is a chronic inflammatory disorder of the airways characterized by recurrent episodes of variable expiratory airflow limitation. Asthmatic patients have hyperresponsive airways that constrict when exposed to various stimuli. Symptoms and airflow limitation are often reversible, either spontaneously or with treatment. However, reversibility may be incomplete in some patients. Although patients appear to recover completely clinically, some asthmatic patients may have chronic airflow limitation. The diagnosis of asthma should be based on the history of characteristic symptom patterns and evidence of variable airflow limitation documented by bronchodilator reversibility testing or other tests.

## Pathophysiology

The mediators released in response to allergens and nonallergic stimuli cause inflammation, edema, mucus production, and airway smooth muscle hypertrophy. All of these lead to bronchoconstriction and hyperreactivity, aggravating airway

obstruction and airflow limitation. The repetitive airway inflammation leads to persistent structural changes in airways, called airway remodeling. This results in increased airway resistance and a decrease in forced expiratory volumes and flow rates. As a result, the lungs become hyperinflated. Ventilation-perfusion mismatch develops despite increased work of breathing. The interaction of these features determines the clinical manifestations, the severity of asthma and the response to therapy. Watch this [video](#).

## Presenting Signs and Symptoms

The classic symptoms include the triad of dyspnea, wheezing and coughing. Physical findings during an asthma exacerbation can be variable. A patient with a mild exacerbation may merely be coughing and complaining of chest tightness, whereas a patient with a severe exacerbation will be in respiratory distress, with tachypnea and loud wheezing. At the other end of the

spectrum are patients with a “silent chest,” which reflects very severe airflow obstruction and air movement insufficient to promote a wheeze.

The exacerbation begins with coughing and a sensation of chest constriction. As the attack advances, expiration is prolonged, wheezing becomes prominent, and accessory respiratory muscles are used. To decrease the effort of breathing, the patient may sit upright or lean forward. The appearance of paradoxical respirations reflects impending ventilatory failure. Alteration in mental status heralds respiratory arrest.

## Critical Bedside Actions

1. Ensure adequate oxygenation
2. Give SABA to reverse airflow obstruction
3. Give systemic corticosteroids to relieve inflammation
4. Carry out serial assessments to monitor the response to therapy

## General Approach

- Obtain patient history, assess exacerbation severity and initiate treatment simultaneously. Respiratory rate, dyspnea, pulse rate, oxygen saturation and lung function reflect exacerbation severity.
- Keep alternative causes of breathlessness in mind.
- Start treatment with repeated doses of inhaled SABA by a puffer, spacer, or nebulizer. Give early oral corticosteroids and controlled flow oxygen in life-threatening exacerbations or if Forced Expiratory Volume in 1 Second (FEV1) is less than 30% of the predicted. Target oxygen saturation is 93-95% in adults and adolescents, and 94-98% in children between 6-11 years.
- Monitor symptoms and oxygen saturation frequently or continuously. Measure lung function after one hour.<sup>3</sup>
- Add ipratropium bromide to treatment in severe exacerbations. Consider

intravenous magnesium sulfate if the patient is unresponsive to intensive initial treatment.

- Do not routinely perform chest x-ray or blood gases, or prescribe antibiotics.
- Prescribe ongoing controller treatment before discharge to reduce the risk of future exacerbations. Provide follow up for all patients, preferably within a week.

## Differential Diagnosis

Wheezing, coughing, and dyspnea may be caused by many other conditions, including pneumonia, bronchitis, croup, bronchiolitis, chronic obstructive pulmonary disease, congestive heart failure, valvular heart diseases, pulmonary embolism, allergic reactions, gastroesophageal reflux disease, exposure to odors, dust, and gas, and upper airway obstruction from vocal cord dysfunction, edema, neoplasm or a foreign body. Any of these alternative diagnoses may also be found together with asthma.

## History/Physical Examination Hints

### History

- Possible causes of the current exacerbation
- The severity of symptoms compared with previous exacerbations
- Other comorbidities
- Current asthma medications and adherence to therapy
- Any use of potential asthma triggering medication
  - Aspirin
  - Beta-blockers
  - Angiotensin-converting enzyme inhibitors
- Any risk factors for asthma-related death
  - poor adherence to asthma medications

- psychosocial problems
- history of near-fatal asthma requiring intubation and mechanical ventilation
- hospital visit for asthma in the past year
- currently using or having recently stopped using oral corticosteroids
- not currently using inhaled corticosteroids
- over-use of SABAs
- food allergies

## Physical Examination

- Check vital signs
- Look for signs of exacerbation severity
  - use of accessory muscles
  - mentation
  - sitting position
  - level of consciousness
- Look for complicating factors

- Anaphylaxis
- Pneumonia
- Pneumothorax
- Look for signs of alternative conditions that could explain acute dyspnea
  - cardiac failure
  - pulmonary embolism
- PEF in patients older than 5 years

The most common finding on physical examination is expiratory wheezing. With severe airway obstruction, it decreases or vanishes because air movement velocity is insufficient to produce sound. Crackles and inspiratory wheezing are not features of asthma. They are more likely to be seen in pneumonia. Inspiratory and expiratory crackles, however, are seen in pulmonary edema.

Patients with the mild or moderate attack can talk in full sentences or phrases. Patients may prefer sitting to lying, but accessory muscles are not used.

Respiratory rate is increased, and the pulse rate is around 100-120 bpm. Oxygen saturation on room air is 90-95%, and PEF > 50% predicted or best.

Patients with a severe attack can only talk in words, sit hunched forwards, use accessory muscles and show agitation. Respiratory rate is >30/min. Pulse rate exceeds 120 bpm. Tachypnea and tachycardia are associated with severe obstruction, but a lower rate does not rule out severe asthma. Oxygen saturation on air is <90% and PEF ≤ 50% predicted or best.

Patients with the life-threatening attack are drowsy or confused and have silent chest.

## Emergency Diagnostic Tests And Interpretation

- Pulmonary Function Tests: Bedside spirometry is used both for initial assessment and for evaluating the response to therapy. If possible, and without delaying treatment, record PEF or FEV1 before treatment. Patient

cooperation is essential for these tests to be reliable. Monitor lung function at intervals until a clear response to treatment has occurred, or a plateau is reached. When possible, management decisions should be guided by the patient's personal best PEF or FEV1 value or, if unknown, predicted values.

- Pulse oximetry: is a noninvasive, convenient and continuous method for monitoring oxygen saturation before and during treatment.
- Arterial Blood Gas (ABG) analysis: is helpful if there is a concern for hypoventilation with carbon dioxide retention and respiratory acidosis. It is not indicated in the majority of patients with mild to moderate asthma exacerbation. Consider ABG analysis for patients with a PEF or FEV1 <50% of the predicted, or for those who do not respond to initial treatment or continue deteriorating despite treatment. A PaO2 <60 mmHg and normal or increased paCO2 (especially

>45 mmHg) indicate respiratory failure. Fatigue and somnolence suggest that pCO2 may be increasing and airway intervention may be needed. Do not wait for arterial blood gas confirmation treat ventilatory or respiratory failure.

- Other Blood Testing: Laboratory studies are rarely helpful in an acute asthma attack.
- Radiology Studies: Radiography is only indicated if there is the possibility of pneumothorax, pneumomediastinum, pneumonia, or other medical conditions.
- Electrocardiogram and Cardiac Monitoring: A routine electrocardiogram is unnecessary. Older patients and patients with coexistent heart disease or with severe exacerbation should undergo continuous cardiac monitoring to detect dysrhythmias.

## Treatment Options

- Oxygen: Administer by nasal cannula or mask. Target arterial oxygen saturation

of 93-95% (94-98% for children 6-11 years).

- Inhaled short-acting beta2-agonists (SABAs): Use 4-10 puffs pMDI with a spacer in mild or moderate attacks. For severe attacks, administer 1 nebulizer every 20 minutes for 1 hour.
- Epinephrine (for anaphylaxis): Indicated only if acute asthma is associated with anaphylaxis and angioedema.
- Systemic corticosteroids: enhance the resolution of exacerbations and prevent recurrence. They should be utilized within 1 hour of presentation. Oral and intravenous administrations are equally effective. However, the oral route is preferred because it is less invasive and less expensive. Intravenous corticosteroids can be administered when patients are too dyspneic to swallow if the patient is vomiting, or when patients require non-invasive ventilation or intubation. Corticosteroid dose is 1 mg/kg (max. 50 mg)



prednisolone for adults and 1-2 mg/kg (max. 40 mg) for children.

- Inhaled corticosteroids: are well tolerated. However, the cost is a limiting factor. The effectiveness, dose, and duration of treatment in the ED remain unclear.
- Ipratropium bromide: Use for moderate-severe exacerbations, along with SABA.
- Magnesium: Intravenous magnesium sulfate is not recommended for routine use in asthma exacerbations. It reduces hospital admissions in patients who fail to respond to initial treatment and have persistent hypoxemia.
- Helium-oxygen therapy: May be considered for patients who do not respond to standard therapy.
- Non-Invasive Mechanical Ventilation (NIMV) and Intubation: If the patient begins to exhibit signs of acute respiratory failure including progressive hypercapnia and acidosis, intubation and mechanical ventilation is indicated.

NIMV is still controversial in asthma. It improves work of breathing, gas exchange. However, increase the risk of barotrauma.

This [video](#) demonstrates treatment of asthma. However, in the ER, the actions should be a lot faster.

## Special Populations

### Pregnancy

The advantages of actively treating asthma in pregnancy markedly outweigh any potential risks of the usual controller and reliever medications. To avoid fetal hypoxia, acute asthma exacerbations during pregnancy should be aggressively treated with SABA, oxygen, and administration of systemic corticosteroids.

### Elderly

The elderly may not describe asthma symptoms or may associate breathlessness with their age or comorbidities (cardiovascular disease, obesity, etc.). The impact of

comorbidities, concurrent treatments, medication side effects (cardiotoxicity with beta2-agonists; skin bruising, osteoporosis, cataracts with corticosteroids) and lack of self-management skills should be taken into account while managing asthma in the elderly.

### Children

Management of asthma exacerbation for adults and children >5 years are mostly similar. This section points to the management of asthma exacerbations in children 5 years and younger.

The presence of any one of these features means a severe exacerbation:

- altered consciousness
- oxygen saturation from pulse oximetry of <92% on presentation
- central cyanosis
- silent chest

- impaired mentation (the normal developmental capability of the child must be taken into account)
- pulse rate (>200 beats/min for children 0-3 years, >180 beats/min for children 4-5 years).

Oxygen: target oxygen saturation is 94-98%.

Bronchodilator therapy: Give 2-6 puffs of salbutamol by a spacer, or 2.5mg salbutamol by nebulizer, every 20 min for the first hour, then reassess severity. If symptoms persist or recur, give an additional 2-3 puffs per hour. For children with moderate-severe exacerbations and a poor response to initial SABA, ipratropium bromide may be added, as 2 puffs of 80 mcg (or 250mcg by nebulizer) every 20 minutes for 1 hour only.

Systemic corticosteroids: Systemic corticosteroids: Give initial dose of oral prednisolone (1-2 mg/kg up to a maximum 20 mg for children <2 years old; 30 mg for children 2-5 years, OR,

intravenous methylprednisolone 1 mg/kg 6-hourly.

Magnesium sulfate: If the child is not responding to standard therapy, consider nebulized isotonic magnesium sulfate (150mg) 3 doses in the first hour of treatment OR intravenous magnesium sulfate (in a single dose of 40-50- mg/kg (max 2g) by slow infusion (20-60 min) for children aged  $\geq 2$  years with severe exacerbation.

## Disposition Decisions

If pre-treatment FEV1 or PEF is <25% of the predicted or personal best, or post-treatment FEV1 or PEF is <40% of the predicted or personal best, hospitalization is recommended.

If post-treatment lung function is >60% of the predicted or personal best, discharge is recommended after considering risk factors and availability of follow-up care.

Patients with post-treatment lung function 40-60% of the predicted are the gray zone. Hospitalization or discharge

decision should be made according to the patient's risk factors and the availability of follow-up care.

Risk factors associated with the need for admission:

- Female sex, older age, and non-white race
- Use of more than 8 beta2-agonist puffs in the previous 24 hours
- Severity of the exacerbation (e.g.need for resuscitation or rapid medical intervention on arrival, respiratory rate >22 breaths/minute, oxygen saturation <95%, final PEF <50% predicted).
- History of severe exacerbations requiring admission to hospital
- Previous healthcare facility visits requiring the use of oral corticosteroids.

An asthma exacerbation does not resolve completely on discharge; airway inflammation and peripheral obstruction may take hours to days to dissipate.

- Prescribe at least a 5-7 day course of oral corticosteroids (prednisolone or equivalent 1 mg/kg/day to a maximum of 50 mg/day), along with inhaled corticosteroids and reliever medication.
- Implement strategies to reduce modifiable risk factors (irritant or allergen exposure, incorrect inhaler skills, inadequate long-term treatment, problems with adherence, or lack of a written asthma action plan).
- Arrange follow-up appointment within one week

**References and Further Reading**, click [here](#)

# Chronic Obstructive Pulmonary Disease (COPD)

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by Ramin Tabatabai, David Hoffman, and Tiffany Abramson

## Case Presentation

*A 68-year-old male presents to the emergency department (ED) with audible wheezing, and he is in severe respiratory distress. He is speaking in 2-3 word sentences, and he is diaphoretic and slightly confused. Per the paramedic report, the patient is a two pack per day smoker. On physical examination, the patient demonstrates poor air movement, and you note that he has a “barrel chest.” As you pick up the phone to call the respiratory therapist for airway management, you wonder, “What other interventions should I initiate and are there other diagnoses I should be considering?”*



Audio is available [here](#)

## General Approach and Critical Bedside Actions

Although COPD patients may frequently visit ED, some of these presentations may require critical interventions such as intubation. Therefore, the ABC sequence should be followed in all these cases to understand an immediate life-threatening situation.

The most patients require Oxygen therapy to keep pulse oximetry 88-92%. Establishment of intravenous (IV) line and fluid replacement may be necessary for severe attacks. Cardiac monitor and electrocardiogram (ECG) to assess cardiac ischemia or arrhythmia is mandatory for every case. While these activities are going on simultaneously, the emergency physicians' primary role is to rule out other life-threatening causes of dyspnea. Inhaled beta-agonist bronchodilator (e.g., Albuterol), Inhaled anticholinergic bronchodilator (e.g., Ipratropium), and oral glucocorticoid therapy (IV steroids only if unable to tolerate PO) are the mainstay of the

treatment in the majority of the patients. BiPAP therapy for moderate to severe exacerbations should be kept in mind. Antibiotic therapy should be started for any acute exacerbation requiring admission or discharged patients with increased sputum purulence.

## Differential Diagnoses

During the initial evaluation and ongoing bedside treatments, emergency physician lists causes of this attack in his/her mind. Two major challenges exist in evaluating the patient with suspected COPD. First, the differential diagnosis for dyspnea is broad and distinguishing COPD from alternative causes can be difficult. Second, patients with COPD may harbor concomitant cardiopulmonary disease.

COPD should be considered in anyone with risk factors and dyspnea, chronic cough or sputum production. Major risk factors include smoking and environmental exposures. Pathological changes that occur in the lung causes air trapping and progressive airflow

limitation. COPD is, therefore, a chronic, progressive disease, usually with an indolent course of gradual decline in airflow and physical activity level secondary to dyspnea.

The etiologies of acute exacerbation can be classified into four different groups (infectious, pollution, destabilizers, idiopathic). Although approximately 70% of exacerbations are due to infection (Viral or Bacterial), it is important to consider other potential triggers or etiologies such as Pneumothorax, Pulmonary Embolism (PE), Congestive Heart Failure (CHF), Pneumonia, Pericardial Effusion, Lobar Atelectasis, Anaphylaxis, Airway Obstruction, and Trauma.

Acute exacerbation of COPD is often confused with pulmonary edema secondary to CHF. Cardiac "wheeze" is easily mistaken for the wheeze classically heard in acute COPD exacerbation. Further complicating matters, these diagnoses are not mutually exclusive and



can often present together in a mixed picture. A thorough evaluation of clinical evidence of CHF is therefore critical in the evaluation of the wheezing acute COPD exacerbation patient.

Additional diagnoses should be considered when an acute COPD exacerbation is more severe than previous or if the patient deteriorates rapidly. One such disease is PE, which can occur in COPD patients due to sedentary lifestyles, increased venous stasis, and increased blood viscosity. Pneumothorax is another critical consideration as COPD patients. As a traditional knowledge, COPD patients have increased risk for ruptured bullae. Other lethal causes of exacerbation and dyspnea are not limited to but include pneumonia and lobar atelectasis secondary to bronchial plugging.

## History and Physical Examination Hints

In the ED, providers are predominately concerned with acute COPD exacerbation. An exacerbation is defined as an acute event that leads to a worsening of the patient's respiratory symptoms, beyond normal day-to-day variation and leads to a change in medication.

The physician's first action in the evaluation of a dyspneic patient with suspected acute COPD exacerbation is airway, breathing and circulation, and assessment of vital sign abnormalities. These are used to determine whether the patient will require immediate intervention. Any of the following signs on initial visual inspection indicate severe acute COPD exacerbation: "tripoding," inability to

speak in full sentences, confusion, agitation, use of accessory respiratory muscles, paradoxical chest wall movements, worsening or new onset central cyanosis, development of peripheral edema, or hemodynamic instability.

A thorough examination will involve cardiopulmonary evaluation to assess for the presence of wheeze and auscultation to estimate the degree of tidal volume that occurs with each ventilation. Markedly decreased air movement indicates severe disease. Other findings in chronic COPD may include a thin, barrel-chested appearance or plethoric, cyanotic appearance.

One important sequela of COPD is cor pulmonale. Long-standing increased pulmonary pressures can lead to right-heart strain and eventual right heart failure. Patients can therefore present with acute COPD exacerbation along with CHF findings of jugular venous distention and peripheral edema.

Finally, a thorough history should be obtained by evaluating risk factors, previous exacerbations, the frequency of exacerbations, and prior intubations. While there are many predictors of a COPD exacerbation, the best is a history of prior exacerbations.

## Emergency Diagnostic Tests and Interpretation

Every patient in respiratory distress should be placed on continuous pulse-oximetry and cardiac monitoring.

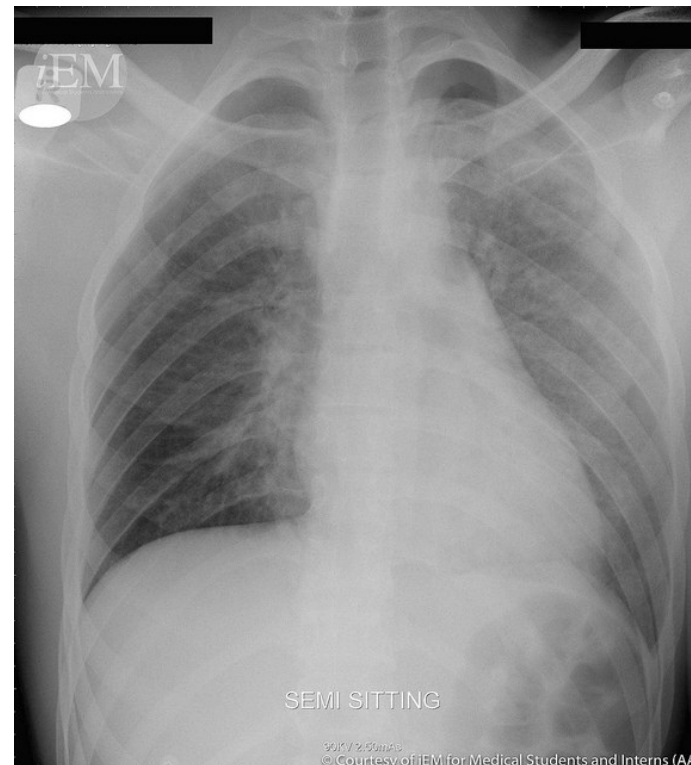
### Bedside Tests

An electrocardiogram is useful in identifying classic patterns (cor pulmonale and dysrhythmias) associated with COPD as well as evaluating for ischemia.

The chest radiograph (CXR) should be ordered to evaluate the presence of other treatable diagnoses. CXR findings in COPD may include a small cardiac silhouette, hyperinflated lung fields, or bullae. Alternative findings may consist of an enlarged heart, effusion or parenchymal consolidation.

What is your diagnosis in the given Chest X-ray of a dyspneic patient?

Image 10.1



Ultrasound should be used when available for the rapid information it provides. A cardiac and lung ultrasound can help both establish and rule out diagnoses by evaluating for pericardial effusion, cardiac squeeze, B-lines, and lung sliding.

The US **video** shows A (normal) and B-lines in the lung.

It is important to note that while spirometry is essential to the formal

diagnosis of COPD in the outpatient setting, there is no role for its use in the emergency room.

### Laboratory Tests

Blood tests have little utility because their results do not change treatment or disposition. The argument can be made for the measurement of brain natriuretic peptide (BNP), which reflects the stretching of myocardial tissue and can, therefore, indicate decompensated heart failure.

## Emergency Treatment Options

### Bedside Critical Actions and Stabilization

The emergency physician must first decide whether the patient requires respiratory assistance. Oxygenation and ventilatory support are mainstay therapies. Patients with hypoxemia need supplemental oxygen with a targeted oxygen saturation goal of 88-92%. This target has been set because high flow oxygen has been associated with carbon

dioxide retention, hypercapnia, respiratory acidosis and respiratory failure. Oxygen delivery is dependent on the severity of the patient's respiratory status and mentation, ranging from the nasal cannula to mechanical ventilation. Noninvasive positive pressure ventilation via BiPAP is an effective treatment for patients in moderate to severe respiratory distress. It decreases treatment failure, reduces complications, shortens hospitalizations, and improves mortality rate. Specifically, a Cochrane review in 2004, demonstrated the use of BiPAP led to decreased mortality (number need to treat, NNT=10), reduction in treatment failure (NNT=5) and decreased need for intubation (NNT=4). Patients with BiPAP failure, however, require intubation and mechanical ventilation. Once intubated, ventilator management and strategy should focus on a prolonged Inspiration and Expiration ratio and low respiratory rate with small tidal volume.

For patients with a high suspicion for acute COPD exacerbation, empiric

treatment with beta-agonist bronchodilators (e.g., albuterol) and anticholinergics (e.g., ipratropium) is generally safe and is considered 1st line therapy. Efficacy between MDI and nebulizer is equivalent, however moderate to severe patients may be unable to use MDI. Nebulized dosing for albuterol should be 2.5 to 5mg and 0.5mg for ipratropium. Combination of both medications is synergistic and relatively safe; however, caution is advised in patients with cardiac disease.

Corticosteroids are the other first-line treatment in acute COPD exacerbation. Studies have demonstrated shorter recovery time, length of stay in the hospital and an NNT=10 to avoid treatment failure. Oral and IV glucocorticoids have similar efficacy, with a recommended dose of prednisone 40 mg PO and methylprednisolone 1-2mg/kg IV. A 5-day course is sufficient and preferable to the side effects caused by longer regimens.

Antibiotic administration is controversial. To simplify this debate, antibiotics for patients with both increased sputum purulence and dyspnea or those requiring hospitalization are recommended. The antibiotic choice should be selected based on the suspected pathogen and whether the patient has risk factors for *Pseudomonas*. First line outpatient antibiotic choices include doxycycline, a beta-lactam, and sulfamethoxazole-trimethoprim (for 5-7 days) and for hospitalized patients contains fluoroquinolones (e.g., levofloxacin) or a beta-lactam with *pseudomonas* coverage (e.g., cefepime).

## **Pediatric, Geriatric, Pregnant Patient and Other Considerations**

COPD is a chronic disease with a peak incidence in the 5th to 6th decade of life. It has been linked to many comorbid conditions making the geriatric population high-risk. Additionally, studies have demonstrated that morbidity in COPD increases with age. Providers,

**References and Further Reading,**  
click [here](#)

should, therefore, demonstrate greater caution with geriatric patients and have a lower threshold to admit.

COPD is not a well-defined disease in the pediatric population and is extremely rare in pregnant patients. Even children with  $\alpha$ 1-antitrypsin deficiency do not develop symptoms until 20-50 years of age. Interestingly, however, the diagnosis of “asthma” in childhood, has been associated with a 10-fold risk for COPD in the future and there is mounting evidence that genetics has a considerable role to play in the development of the disease.

## Disposition Decisions

Criteria for hospitalization include an exacerbation failing to improve back to baseline. The GOLD collaborators have outlined the criteria for admission and discharge and further delineated criteria for ICU admission versus ward (Table). Criteria for ICU admission revolve around respiratory failure and altered mental status.

**Image 10.2** COPD Admission and Discharge Criteria (GOLD)

Admission Criteria
Ward
Failure to respond to initial medical management
A marked increase in the intensity of symptoms
Severe underlying COPD, severe comorbidities or old age
Insufficient home resources
ICU
Persistent or worsening hypoxia despite supplemental oxygen
Need for invasive mechanical ventilation
Change in mental status
Hemodynamic Instability
Discharge Criteria
Clinically stable
The frequency of medication use <4hrs
Dyspnea does not negatively effect sleeping, eating or basic functions
Able to complete ADLs and understands medications

*Adapted from the Global Strategy for the Diagnosis, Management, and Prevention of COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2015. Available from: [http://www. goldcopd.org/](http://www.goldcopd.org/).*

# Pneumonia

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by Mary J. O.

## Case Presentation

*A 74-year-old male with a history of hypertension and diabetes presented to the emergency department with a cough productive of rust-colored sputum. His complaints started approximately three days earlier and progressively worsened. The patient reported difficulty in breathing, shaking chills, and fever up to 39°C. He had no sick contacts. On examination, the physician noticed an elderly gentleman in mild respiratory distress. His vital signs were: BP: 110/70 mmHg, HR: 102 bpm, RR 20 bpm, T 38.4°C and SpO2 91% on room air. Auscultation revealed rales at the right lung base.*



## Introduction

Pneumonia is an acute respiratory infection of the lung parenchyma, particularly the alveoli. The healthy alveoli fill with air when a person breathes; however, in pneumonia, the fluid or pus in the alveoli makes breathing painful and inhibits air exchange. Despite modern research and the development of a variety of antimicrobial agents, pneumonia remains a leading cause of death worldwide, especially in the very young and the elderly.

The most common causes of pneumonia are bacteria and viruses, but fungi, protozoans, and parasites can also cause infection. These organisms, typically found in the nasopharynx, can infect the lungs by inhalation. Additionally, airborne droplets (such as from a cough or sneeze) or blood-borne infections (such as from mother to baby during delivery) may spread the disease.

For an overview of pneumonia, watch this [video](#).

## Classification

Community-acquired pneumonia (CAP) occurs in patients with no recent hospitalization or exposure to the healthcare system. The most common bacterial cause of CAP is *Streptococcus pneumoniae*. Its incidence is declining due to vaccination. Other common bacteria are *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Staphylococcus aureus*. These so-called “typical” pneumonia agents generally present with primarily respiratory symptoms and a lobar consolidation on chest radiograph. *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, and *Legionella* sp. are among the common atypical causes of bacterial pneumonia. They may present with a subacute onset, more generalized, non-respiratory symptoms and respond to different antibiotics than the typical organisms. The radiographs may not show an infiltration.

Common viral causes of pneumonia include influenza, respiratory syncytial

virus (RSV), parainfluenza, coronaviruses, adenoviruses, and rhinoviruses. Recently, a number of new viral pathogens have emerged, including coronaviruses that causes severe acute respiratory syndrome (SARS) and the Middle East respiratory syndrome (MERS-CoV).

Hospital-acquired pneumonia (HAP) refers to pneumonia newly-contracted at least 48-72 hours after hospitalization. It is the second most common type of nosocomial infection (after urinary tract infections), and a common cause of death in the intensive care unit. Ventilator-associated pneumonia (VAP) is pneumonia that occurs 48 hours or more after a patient receives mechanical ventilation through an endotracheal tube or tracheostomy. Intubation allows oral and gastric secretions and microorganisms to enter the lower respiratory tract. Multidrug-resistant (MDR) organisms usually cause VAP.

Healthcare-associated pneumonia (HCAP) is a particular subset of nosocomial pneumonia in which patients come from the community but have frequent interactions with the healthcare system. It includes patients who were hospitalized within the last 90 days, reside in a long-term care facility such as a nursing home, receive hemodialysis or wound care, have contact with a family member with MDR pathogens, or are on chemotherapy or intravenous antibiotics. Like HAP and VAP, patients with HCAP are at risk for multidrug-resistant pathogens.

## Critical Bedside Actions and General Approach

Initial evaluation should focus on ensuring adequate ventilation and oxygenation. Hypoxic patients should receive supplemental oxygen. Endotracheal intubation may be required in patients with severe respiratory distress. Early and aggressive fluid resuscitation is necessary for patients who are hemodynamically unstable or who are

presenting with sepsis. Empiric antibiotic therapy should be started once the diagnosis of pneumonia is established, even before the definite identification of a microbial cause.

## Differential Diagnosis

- Asthma
- Bronchitis
- Chronic obstructive pulmonary disease (COPD)
- Lung cancer
- Pulmonary edema
- Pulmonary embolism
- Upper respiratory tract infections

## Clinical Presentation

The classic symptoms of pneumonia are fever/chills, cough (often productive of purulent sputum), pleuritic chest pain, and shortness of breath. Elderly patients may present with nonspecific symptoms, such as general malaise, anorexia, and confusion. On physical examination,

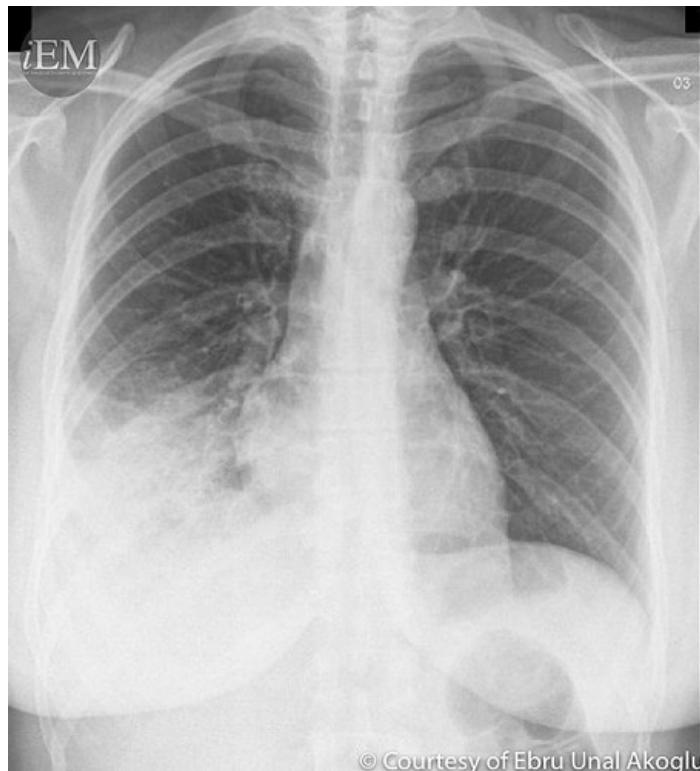
tachycardia and tachypnea are usually present. Patients may be hypoxic and hypotensive. Auscultation of the chest may reveal coarse rales or bronchial breath sounds. There may also be dullness to percussion and increased tactile fremitus. No single clinical finding is reliable in establishing a diagnosis of pneumonia.

For examples of lung sounds, please see the this [video](#).

## Emergency/Diagnostic Tests and Interpretation

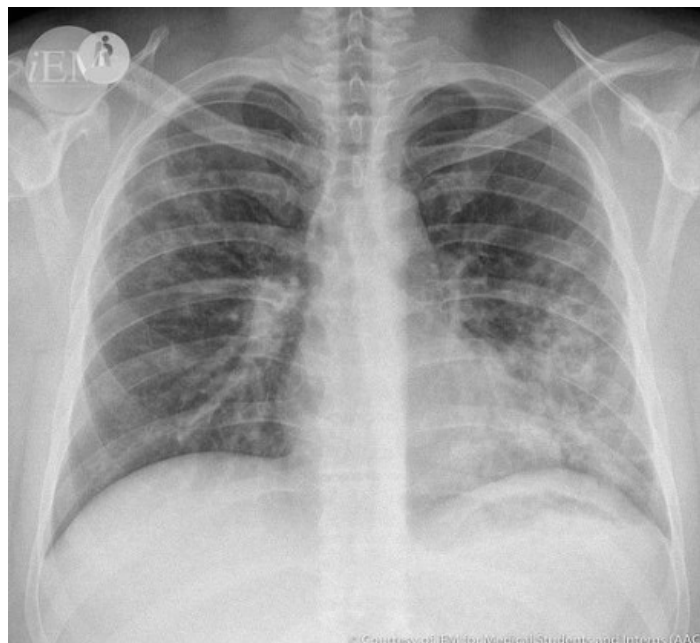
- Pulse oximetry to screen for hypoxia. Hypoxia is an indication for admission.
- Chest x-ray (CXR): generally the most important study to determine the presence of pneumonia, although it cannot establish the causative agent. The absence of findings on CXR should not preclude the use of antibiotics in patients thought to have pneumonia based on clinical presentation.

**Image 10.3**



- Computed tomography (CT) of the chest: more sensitive than CXR, but often not necessary.
- Point-of-care ultrasound is becoming more widely used for the rapid diagnosis of pneumonia. It can be more sensitive than CXR, though findings of consolidation on ultrasound are not specific for pneumonia. The accuracy of ultrasound is operator-dependent.
  - This ultrasound **video** shows consolidation and pleural effusion related to pneumonia.

**Image 10.4**



- A complete blood count (CBC) may show the presence of leukocytosis, but this often does not affect overall management.
- An elevated lactate level may indicate the need for more aggressive fluid resuscitation.
- If congestive heart failure is in the differential diagnosis, B-type natriuretic

peptide (BNP) may help distinguish between CHF and pneumonia.

- The utility of routine blood cultures has been questioned due to the low yield, but they should be drawn before the initiation of antibiotics in patients who will be admitted, particularly in severely ill patients. Cultures are positive in 20-25% of pneumonia caused by *S. pneumoniae*, but the percentage is even lower in pneumonia due to other causes. Positive blood cultures may help determine local antibiotic resistance patterns.
- Sputum Gram stain and culture can help identify a bacterial pathogen, but like blood cultures, the usefulness in the emergency department setting is very limited. With an adequate specimen, sputum studies are positive in more than 80% of cases of *S. pneumoniae*, but that percentage is much less for other pathogens.

- Urine antigen tests are available for *Legionella pneumophila* serotype 1 (74% sensitivity) and pneumococcus.
- Rapid diagnostic tests are available for many viruses, including RSV and influenza.

## Treatment Options

Once pneumonia has been diagnosed, antimicrobial therapy should be started as soon as possible, as early initiation leads to better outcomes. Antimicrobial therapy should be tailored to the most likely causative organisms in order to avoid drug toxicity, decrease the rate of resistance to broad-spectrum antibiotics, and reduce cost. The empiric treatment of CAP has been made more difficult by the emergence of drug-resistant *Streptococcus pneumoniae* (DRSP).

Risk factors for resistant *S. pneumoniae* include age > 65 years; recent treatment or repeated therapy with beta-lactams, macrolides, or fluoroquinolones; and medical comorbidities, including immunosuppression.

The following recommendations are adapted from: the EMRA Antibiotic Guide; Musher et al., NEJM; and World Health Organization (WHO) guidelines.

### Outpatient therapy (adults):

- Amoxicillin/clavulanate, with the addition of azithromycin or doxycycline if atypical organisms are suspected
- Levofloxacin or moxifloxacin monotherapy may be used instead
- If influenza is suspected, treat early with oseltamivir

### Inpatient therapy (adults):

- Ceftriaxone or cefotaxime, with the addition of azithromycin or doxycycline
- Levofloxacin or moxifloxacin may be used instead
- If influenza is suspected, treat early with oseltamivir
- If MRSA is suspected, vancomycin or linezolid should be added

- If the patient is allergic to penicillin, use aztreonam and levofloxacin instead
- If *Pseudomonas* is likely, use double coverage until susceptibilities are back – levofloxacin or gentamicin, with the addition of an anti-pseudomonal beta-lactam: cefepime, piperacillin/tazobactam, or aztreonam

### HCAP, HAP, or VAP (adults) [choose one from each category below]:

- Beta-lactams: cefepime, ceftazidime, piperacillin/tazobactam, aztreonam
- Levofloxacin, or azithromycin plus gentamicin
- MRSA coverage: vancomycin or linezolid

### Outpatient therapy (pediatrics):

- Amoxicillin (preferred in low-resource settings) or amoxicillin/clavulanate
- If atypical pneumonia suspected, azithromycin



## Inpatient therapy (pediatrics):

- Ampicillin
- Ceftriaxone or cefotaxime
- If MRSA is suspected, add clindamycin or vancomycin
- If atypical organisms suspected, azithromycin

## Special Considerations

### Specific Organisms and Associations

- *S. pneumoniae* is the single most commonly identified organism in both adults and children with CAP. It is classically associated with rust-colored sputum and lobar consolidation.
- *H. influenzae* is a common pathogen in patients with chronic obstructive pulmonary disease (COPD), diabetes, malignancy, alcoholism, and malnutrition.
- *M. catarrhalis* is another common pathogen in patients with COPD,

although it more often causes COPD exacerbations than pneumonia.

- *Klebsiella pneumoniae* rarely causes CAP in a healthy host but can cause severe pneumonia in patients with chronic illnesses, such as alcoholism or diabetes. The sputum produced is often mixed with blood, giving it the color of red currant jelly.
- *S. aureus* can cause severe, necrotizing pneumonia with cavitory lesions. It can often occur after an infection with influenza. Intravenous drug users can also get hematogenous spread.
- *C. pneumoniae* is a relatively common cause of CAP, causing subacute systemic symptoms, such as fever, malaise, myalgias, and non-productive cough.
- *M. pneumoniae* is a common cause of CAP, particularly in younger patients. It can be associated with rashes, sore throat, and ear pain. The classic finding associated with this pathogen is bullous

myringitis, though it is actually not common and is nonspecific.

- The *Legionella* genus is comprised of over 50 species of intracellular organisms that live in aquatic environments and cause infection when inhaled. There have been no documented cases of person-to-person transmission. Elderly patients are at risk of severe pneumonia, often with gastrointestinal symptoms such as abdominal pain and diarrhea. Hyponatremia is often present as well.
- Anaerobic infections can result from the aspiration of oropharyngeal contents. These infections are often polymicrobial. Risk factors are patients with decreased level of consciousness or severe periodontal disease.
- Fungi are a rare cause of pulmonary infections. *Histoplasma capsulatum* lives in soil that contains large amounts of bird or bat droppings and causes disease when spores are inhaled. It is found most commonly in the central



and eastern United States, near the Ohio and Mississippi River valleys. *H. capsulatum* can also be found in Central and South America, Asia, Africa and Australia. *Coccidioides immitis* is found in the southwestern United States and Central and South America.

- Other rare causes of pneumonia are: tularemia (caused by the bacterium *Francisella tularensis*) is spread by contact with infected mammals, especially rabbits. Psittacosis (caused by *Chlamydia psittaci*) can be spread to humans from infected birds. Sheep, cattle, and goats are the natural reservoir of Q fever (caused by *Coxiella burnetii*).

### **Pediatric Patients**

Pneumonia is the leading cause of death of children worldwide, killing more than malaria, measles, and AIDS combined. In neonates, organisms that colonize the maternal vaginal canal, such as Group B *Streptococcus* (GBS), *Listeria monocytogenes*, and *Escherichia coli*

may cause pneumonia. Viruses are a common cause of pneumonia in infants and young children. RSV and parainfluenza infections most often occur in the wintertime. Symptomatic treatment is recommended for cases of viral pneumonia. In school-age children, *M. pneumoniae* and *C. pneumoniae* infections are more common. Pediatric patients often do not present with the classic signs of pneumonia, such as a productive cough. Often, the only signs are tachypnea and increased work of breathing.

### **Geriatric Patients**

Elderly patients are more susceptible to pneumonia than younger patients and have a higher rate of morbidity and mortality. Even after recovery from pneumonia, they have a higher rate of mortality in one year. They often do not present with the classic signs and symptoms of pneumonia, such as fever, cough, sputum production, or leukocytosis. Frequently, the only sign of pneumonia may be confusion or malaise.

The most common cause of community-acquired pneumonia in the elderly remains *S. pneumoniae*. However, geriatric patients are more likely to have resistant types of this organism.

### **Pregnant Patients**

Pneumonia is the most frequent cause of non-obstetric infection in the pregnant patient. The pregnant woman is predisposed to infection due to an alteration in immune status, making her more susceptible to infection. In the postpartum period, pneumonia can occur from aspiration as a complication of obstetric anesthesia. Additionally, there is an increased risk of aspiration due to increased intragastric pressure from the gravid uterus and relaxation of the gastroesophageal sphincter due to progesterone.

Evaluation and treatment are more complicated because there are two patients to care for simultaneously. Chest radiography should be performed with the protection of the fetus. Appropriate

antimicrobial therapy should be selected to avoid teratogenicity. Fetal complications are common as fever and hypoxemia are harmful to development. Preterm labor is a known complication of pneumonia, and tocolytic therapy may be required. Testing for Group B Streptococcus before delivery and intrapartum administration of antibiotics can prevent the transmission of the bacteria to the neonate.

### Immunocompromised Patients

*Pneumocystis jirovecii* (formerly *P. carinii*) is a fungal agent that does not cause infection in healthy people but is an important cause of opportunistic infection in immunocompromised hosts. It remains the most common AIDS-defining illness in individuals with human immunodeficiency virus (HIV). The classic symptoms are fever, nonproductive cough, fatigue, shortness of breath especially with exertion, bilateral interstitial infiltrates, and hypoxia. Trimethoprim/sulfamethoxazole (TMP/SMX), also known as cotrimoxazole, is the drug of

choice for PCP, although alternatives (such as pentamidine, dapsone, and atovaquone) are often needed due to allergic reactions, adverse effects, or treatment failure. Adjunctive therapy with corticosteroids has been shown to improve survival, especially in patients who are hypoxic.

Another pathogen to consider in immunocompromised patients is *Mycobacterium tuberculosis*. Cytomegalovirus (CMV) and varicella zoster are rare causes of viral pneumonia.

### Disposition Decisions

A number of clinical prediction rules and guidelines have been developed to determine whether patients with CAP should be admitted or can be safely treated as an outpatient. As with all clinical prediction rules, these scores should be used as a guideline and should not override the judgment of the physician.

### Pneumonia Severity Index (PSI)

The PSI is the most widely studied clinical prediction rule for pneumonia. It stratifies patients into five classes for risk of death (Risk Class I to V) from all causes within 30 days of presentation based on medical history, physical examination, and laboratory/radiologic findings. All-cause mortality ranges from 0.1% for Risk Class I to 27.0-29.2% for Class V. As points are assigned by age, it may underestimate severe pneumonia in otherwise young, healthy patients and may overestimate severity in older patients (any patient over 50 years of age is automatically classified into Risk Class II).

Click on the [link](#) to access a calculator for **PSI**.

### CURB-65 severity score

The PSI score uses twenty variables and may be cumbersome to use in the emergency department. The CURB-65 score only requires five variables and is easier to compute.

- Confusion

References and Further Reading,  
click [here](#)

- Urea > 7 mmol/L (in the United States, blood urea nitrogen > 19 mg/dL)
- Respiratory rate  $\geq 30$  breaths/minute
- Blood pressure (systolic < 90 mmHg or diastolic  $\leq 60$  mmHg)
- Age  $\geq 65$  years

One point is given for each variable and patients can be stratified according to increasing risk of mortality, ranging from 0.7% mortality for a score of 0 to 57% mortality for a score of 5. Consider an ICU admission for patients with a score of 4 or 5.

Compared to the CURB-65 score, the PSI identified a greater number of patients as low-risk (68% vs. 61%). The low-risk patients according to the PSI had a slightly lower 30-day mortality (1.4%) compared to the CURB-65 (1.7%). The clinical relevance of the slightly improved accuracy of the PSI is unknown.

To calculate a **CURB-65** score, click on the [link](#).

## SMART-COP

The SMART-COP rule is a clinical rule that predicts which patients with community-acquired pneumonia may need intensive care, such as mechanical ventilation or inotropic support. A SMART-COP score of  $\geq 3$  points identified 92% of patients who required intensive care measures. While this score was superior to CURB-65 for predicting whether a patient would need intensive respiratory or vasopressor support, like the PSI, the sensitivity of SMART-COP is reduced in younger patients and was noted in one study to stratify 15% of young adults incorrectly.

To see the **SMART-COP** tool, follow the [link](#).

Ultimately, the decision to admit a patient depends on the physician's judgment, but all the factors listed in the above scoring systems should be considered.

# Spontaneous Pneumothorax

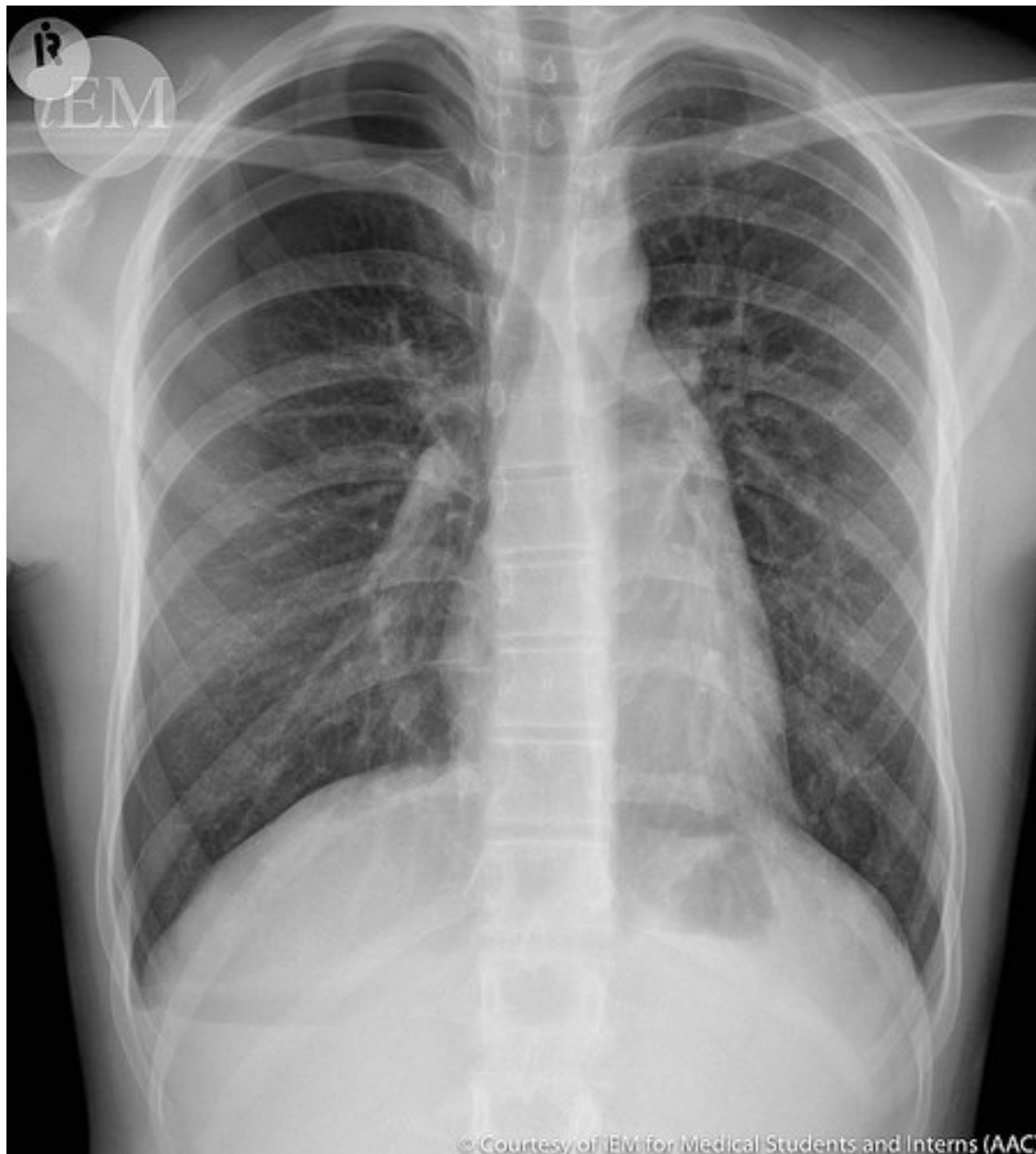
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by Mahmoud Aljufaili

## Case Presentation

*A 26-year-old male, with no significant medical history, presented to the emergency department with acute shortness of breath and associated right-sided chest pain. The pain started suddenly while the patient was at rest, it was sharp and worsening with inspiration. He denied a history of trauma, fever, cough or any other constitutional symptoms. In the ED, apart from tachypnea, his vitals were within normal limits. He was not in distress. The trachea did not deviate. Breath sounds were markedly diminished on the right side, with normal breath sounds on the left side. No wheeze or crackles were appreciated. The chest x-ray is shown below. What is your diagnosis and plan for this case?*

Image 10.5



## Introduction

Pneumothorax refers to the presence of air in the pleural cavity. It can impair oxygenation/ventilation. There are two types of spontaneous pneumothorax 1) primary, and 2) secondary. Primary refers to no underlying disease. Secondary refers to underlying pulmonary disease which has a worse prognosis.

### Causes of secondary pneumothorax are various as follows;

- Vascular: Pulmonary infarct
- Airway disease: Asthma, COPD, Cystic fibrosis
- Neoplasm: Primary or metastatic
- Infective: Tuberculosis. Pneumocystis carinii pneumonia, lung abscess
- Interstitial lung disease: Sarcoidosis, Idiopathic pulmonary fibrosis
- Miscellaneous: Endometriosis

### Risk factors increasing spontaneous pneumothorax risk;

- Family history
- Smoking
- Change in atmosphere pressure



- Marfans – in the absence of lung disease

## Critical Bedside Actions and General Approach

Assess the stability of the patient: Our first responsibility is to evaluate the patient vitals and control to airway, breathing, circulation abnormalities. In any instability, immediate actions are needed to stabilize the problem. Although there is a low chance to have a tension pneumothorax in spontaneous pneumothorax, this can be the worst case scenario for those patients. Oxygen, IV lines and cardiac monitorization may be necessary. Needle or tube thoracostomy can be necessary immediately on the bedside. These critical bedside actions are rarely needed for asymptomatic or mildly symptomatic patients. Therefore, we may have time to use proper diagnostic techniques for differential diagnoses.

Size of pneumothorax: When we diagnosed pneumothorax, the patient

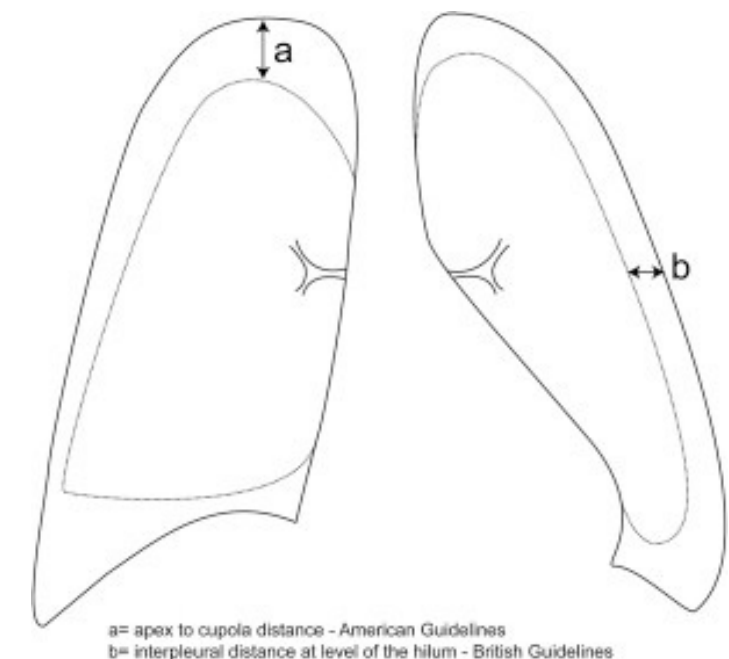
symptoms are the driven factor for the treatment options. However, knowing the pneumothorax size is useful to decide the next step in majority of the cases. At least, today, the algorithms are still designed to the size of the pneumothorax.

How to estimate the size of the pneumothorax?

- On the bedside, this can be done with Chest x-ray. It is ideal to get upright, postero-anterior, and inspirium-expirium x-rays. However, good quality, sitting antero-posterior bedside portable x-rays may guide us well.
- British Thoracic Society guidelines:
  - If the interpleural distance at the level of the hilum is
    - less than 1 cm, then it is a small pneumothorax
    - 1 to 2 cm, then it is a moderate pneumothorax

- more than 2 cm, then it is a large pneumothorax
- The American College of Chest Physicians
  - If the distance from apex to cupola is
    - Less than 3 cm, then it is a small pneumothorax
    - More than 3 cm, then it is a large pneumothorax

**Image 10.6**



MacDuff A, Arnold A, Harvey J Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010 Thorax 2010;65:ii18-ii31.

- Primary or secondary

## Differential Diagnoses

The patients present mostly with shortness of breath (SOB). Therefore, pulmonary, cardiac and other causes of SOB should be considered first.

### Pulmonary

- Airway obstruction
- PE
- Pulmonary edema
- Anaphylaxis
- Asthma
- Cor pulmonale
- Aspiration

### Cardiac

- MI
- Tamponade
- Pericarditis

### Others

- Esophageal rupture
- Toxin ingestion
- Epiglottitis
- Anemia

If the pneumothorax is made clinically or radiologically, then the types should be confirmed as spontaneous, traumatic, primary, secondary or tension etc.

## History and Physical Examination Hints

Sudden pleuritic chest pain. Most often occur at rest.

Increased work of breathing and tachypnea can be seen in moderate and severe pneumothorax.

Hypoxemia can be seen in severe cases. Normal oxygen saturation does not rule out pneumothorax.

Reduced breath sound on the affected side is more obvious with the increased size of pneumothorax (moderate or severe). Auscultation may not appreciate

the difference in small pneumothorax, especially in a busy and noisy ED environment.

Hypotension (think tension pneumothorax!). This is very important “red flag” for a pneumothorax patient. The patients are generally agitated because of hypoxemia and low blood supply to the brain. This finding should warn physicians to immediate action to treat the pneumothorax.

## Emergency Diagnostic Tests and Interpretation

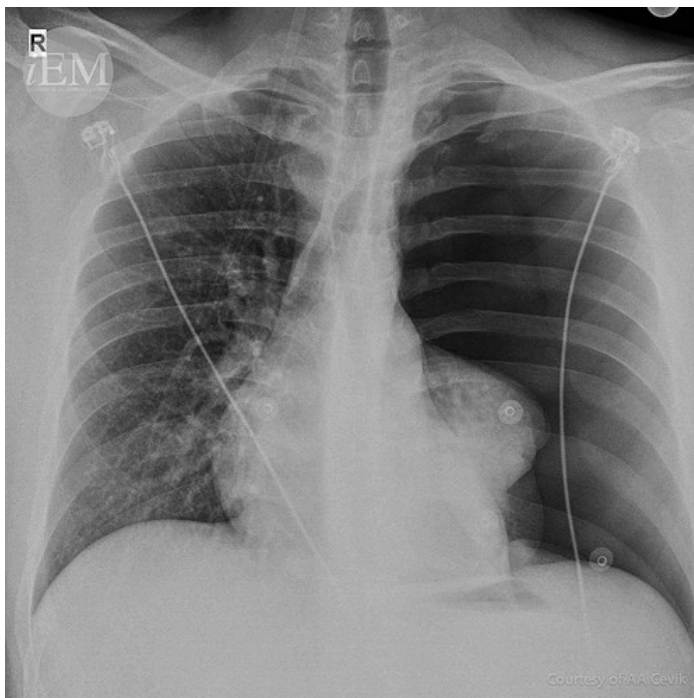
### Chest X-ray

- Displaced visceral pleural line without lung markings between pleural line and chest wall
- Deep sulcus sign on supine x-ray

The Chest x-ray shows left side large pneumothorax with fully collapsed lung tissue. If the patient is vitally unstable (hypotensive, tachycardic, hypoxemic) and agitated, then this x-ray means “tension pneumothorax.” If the patient

vitaly stable, there is no tension. There is very important teaching point for all physicians. tension pneumothorax is a clinical diagnosis, not imaging diagnosis. Therefore, if the patient is clinically unstable and there is no breath sounds on the left side, this is tension pneumothorax until proven otherwise, and this x-ray should not be ordered. Nowadays, yes, you can use ultrasound in seconds to diagnose if you are in doubt.

**Image 10.7**



## Ultrasound

- No sliding lung sign
- Barcode (instead of the wave on the beach) appearance on M-mode ([video](#))

This [video](#) shows normal (left) and abnormal (right) lung findings. Left side shows normal pleura and lung tissue relation and called seashore sign. Right one shows no clear differentiation between these structure and look like a barcode. This finding is a warning for pneumothorax presence.

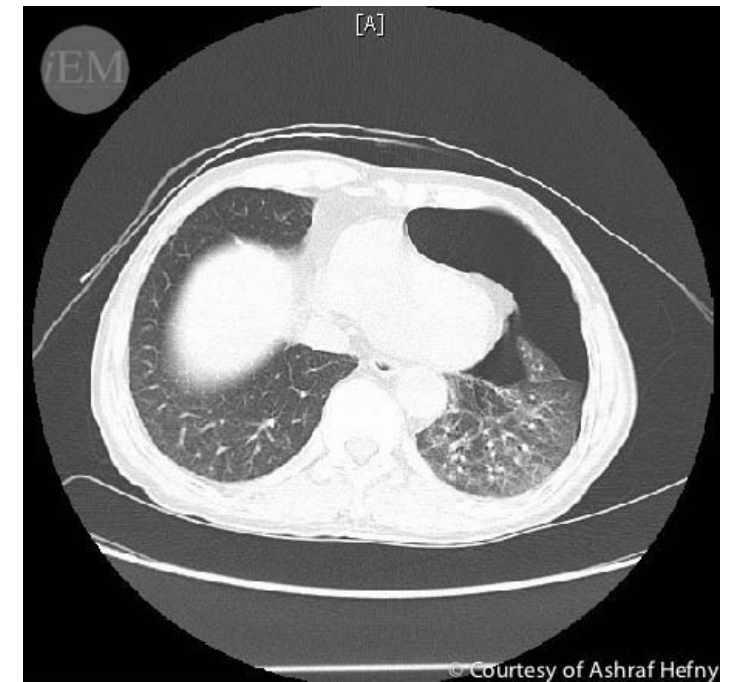
This [video](#) shows “lung Point.” Please pay attention to hyperechogenic (white) line (pleura). Half of the pleura is moving, but other half is standing still (no lung sliding). The connection point is the border of pneumothorax line.

## Computerized Tomography

Very sensitive and specific

CT image shows left side pneumothorax, collapsed lung tissue.

**Image 10.8**



## Emergency Treatment Options

Initial Stabilization with oxygen, needle or tube thoracostomy (if necessary) is the the first priority for unsatble patients.

Oxygen: increase pleural air resolution by 3-4 times, and alone helps to absorbtion of the air by 1-2% a day. Therefore, some institutions may prefer to observe small pneumothoraxes with supplemental oxygen.

## Procedures

Needle or catheter aspiration as effective as chest tube for small pneumothorax. Therefore, they are both appropriate treatment options. Although large pneumothoraxes may require tube thoracostomy, choosing the narrow tube size is effective as wide tubes. Most of the spontaneous pneumothorax are easily and safely treated with pig-tale catheters.

Needle decompression ([video](#)) is necessary for tension pneumothorax. The classical teaching was 2nd intercostal space, mid-clavicular line, over the rib insertion. However, this location is recently controversial, especially in obese patients. Therefore, for adults, the new location is mid-anterior axillary line crossing with 4-5 th intercostal line. This location is also entry side of the chest tube. The below video demonstrates old version of needle decompression. However, this location is still acceptable in skinny adults and children.

This [video](#) demonstrates chest tube insertion

The image shows left side chest tube location.

**Image 10.9**



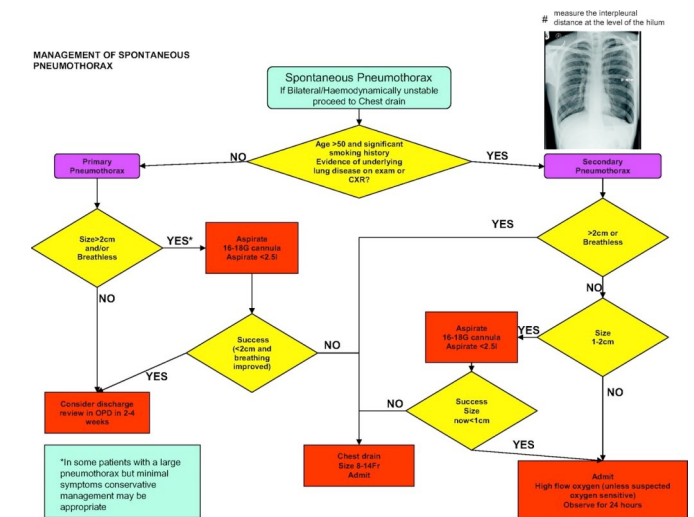
## Disposition Decisions

Recurrent pneumothorax, the patient with abnormal vitals, bilateral pneumothorax, and all secondary pneumothorax should be admitted.

Small pneumothorax with no symptoms and normal findings can be discharged.

Please see the below algorithm below to understand possible treatments and

**Image 10.10** Management of Spontaneous Pneumothorax



MacDuff A, Arnold A, Harvey J Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010 *Thorax* 2010;65:ii18-ii31.

disposition decisions.

If patients were discharged no flying for a week after resolution and no diving are standard recommendations.

**References and Further Reading**, click



## Chapter 11

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# Selected Psychiatric Emergencies





# Acute Psychosis In The Emergency Department

by Elizabeth Bassett, Nidal Moukaddam, and  
Veronica Tucci

## Case Presentation

*A 25-year-old female is brought in by police after being found in a gas station, behaving bizarrely, talking to herself. The patient has no identification, cannot provide her name, and no medical history is available. She is responding to internal stimuli, responds to questions with inappropriate laughter and illogical statements such as “look, a bird, I am queen, meow, what, Jesus, leave me alone,” and not making eye contact with staff. She is noted to be paranoid, repeatedly looking at the air vent above her bed. Initially calm, she became violent after attempts to establish IV access. Verbal de-escalation and redirection were not fruitful. The patient was placed in physical restraints and eventually required emergency pharmacologic intervention. Initial vital signs are Temperature 100.1 Fahrenheit (38.4 degree Celsius), HR 120 bpm, BP 110/75 mmHg, RR 24 per minute, O2 saturation 98%. A liter*



Audio is available [here](#)

*of IV fluids is given, and an hour later the patient is sleeping, physical restraints are removed, and all vital signs are within normal limits.*

*The patient wakes up again, slightly calmer, but is perseverating on being pursued by a dark organization which can read her mind, and wants to cast her in a pornographic video. She insists that the technician assigned to the ED is an agent of evil, and refuses further vital signs. She also refuses oral medications, and when asked if pregnant, lowers her voice and says “that’s why they’re after me, help me please.” She refuses to provide further history, and from that point on, becomes mute.*

## Introduction

Acute psychosis may be encountered on a daily basis in the emergency department (ED). Psychosis is characterized by disorganized thinking, delusions (false, unshakable beliefs), and hallucinations, often auditory, visual. Acute psychosis can also be accompanied by behavioral changes and agitation that are not necessarily commensurate with the severity of psychotic symptoms. The role of the emergency physician, in addition to medically stabilizing and treating acute agitation/psychosis, is to determine whether the patient is experiencing symptoms related to primary psychiatric diagnosis or secondary to a medical illness. This can be challenging given both the limited time and history available to the emergency physician, and the often-noted lack of cooperation of acutely psychotic patients, known to be amongst the hardest amongst patients in the emergency room. The distinction, however, is critical, as incorrectly diagnosing a patient’s behavior as primarily psychiatric in nature, and missing causes of altered mental state (AMS), can lead to dire consequences for the patient. This chapter will cover management aspects of psychosis in the ED.

## General Approach and Critical Bedside Actions

1. Ensure the safety of the patient and the medical staff. Have a low threshold to call security personnel early as unsafe behavior can escalate quickly. Physical restraints and medications may be required early on.

2. Recognize abnormal vital signs.
3. Have the patient put on a telemetry monitor and establish IV access.
4. Assess for immediately life-threatening and reversible causes of psychosis. Check a point of care glucose for hypoglycemia, check oxygen saturation for hypoxemia, and expose the patient to look for evidence of trauma.
5. Perform a head to toe complete physical exam with attention to neurologic exam.

## Differential Diagnosis

1. Primary psychiatric etiology: schizophrenia versus schizoaffective disorder, depression with psychotic features, bipolar disorder with psychotic features
2. Hypoglycemia
3. Drug or alcohol intoxication or withdrawal

4. Substance-induced psychosis (think stimulants, e.g., amphetamines, synthetic cannabinoids)
5. Infection – either systemic or central nervous system (think sepsis or encephalitis)
6. Central nervous system lesion (particularly in patients with a history of cancer or AIDS)
7. Intracranial bleed (especially if evidence of trauma or anticoagulated patient)
8. Hyperthermia/heat stroke (unlikely with a temperature of 100.1, however, checking a rectal temperature may reveal significantly higher core body temperature)
9. Hypoxia/hypercarbia (unlikely given this patient's pulse oximetry reading)
10. Vitamin deficiencies (Wernicke's)
11. Hypotension/hypoperfusion (unlikely with systolic of 110 unless the patient

routinely has markedly elevated blood pressure values)

## 12. Thyrotoxicosis

## History Taking and Physical Examination Hints

### History Taking Hints

A patient with acute psychosis may be a poor historian and collateral information (from police, Emergency Medical Services, or patient's family) is often helpful. Make every effort to meet with them at the time of arrival, as such information may not be available later on. For our patient, no history is available; however, in general, the key information sought includes:

1. Pick up location: Street, home, nursing home? Get contact information of the family or nursing home if available, including prescribed medications. If the patient was from home or nursing home, was any medical history provided such as a history of mental illness or past similar episodes. The

absence of previous psychiatric history strongly suggests a medical cause of the behavior.

2. Timing: When was the patient last seen at baseline? Has the onset of abnormal behavior been gradual or rapid? Rapid onset suggests underlying medical condition or drug use.
3. What did the scene look like: Empty pill bottles, alcohol, illicit drugs, a potential for other toxic exposures? Were there any prescription medications at the scene? Obviously, the presence of prescription anti-psychotics can help with the diagnosis but the entire medication list can help with determining the patient's past medical history and may be the key in diagnosis if the etiology is medical in nature.
4. Vitals and blood sugar of the patient en route to the hospital if available.
5. The mental status of the patient at the scene vs. on arrival. Is there a waxing

and waning course? Is the patient's mental status improving or declining?

6. Has the patient been starving themselves because of psychotic beliefs?
7. How paranoid is the story the patient is telling you? While individuals may indeed be the target of mysterious organizations, most paranoid, persecutory delusions are extremely unlikely.
8. Do they have pre-existing medical conditions they have been neglecting because of their psychosis? Many patients with mental illness have medical comorbidities, often poorly treated.

## Physical Examination Hints

It is imperative that a head to toe exam be performed. This may be the only full physical exam that the patient receives while in the hospital and therefore the only opportunity to assess for traumatic

or medical reasons for the symptoms at hand.

1. HEENT: Look for evidence or recent trauma – lacerations, abrasions, hematoma, basilar skull fracture (raccoon eyes, battles sign, CSF or blood in the ears). Look for evidence of past traumatic brain injury as evidenced by old neurosurgical scars.
2. Eyes: Pay special attention to the ocular exam. Assess for pupil size and responsiveness to light, presence or absence of extra-ocular eye movements, and presence of nystagmus. Ocular findings can be a clue towards various toxidromes or space-occupying lesions.
3. Neck: Assess for meningismus and thyromegaly
4. Pulmonary: The presence of rales, wheezing can be a clue that the patient is experiencing hypoxia secondary to CHF, COPD, or asthma. Hypoxia can be a cause of the patient's altered



mental status although unlikely if the patient's oxygen saturation is high. Rales or diminished breath sounds may be a clue, especially in elderly patients, that the AMS is secondary to a pulmonary infection.

5. Cardiac: This exam is unlikely to aid in the diagnosis; however the presence of an irregularly irregular heartbeat may indicate that the patient is anticoagulated and therefore at increased risk of spontaneous or traumatic intracranial bleed. Tachycardia or bradycardia may also indicate various toxidromes.
6. Abdomen: Assess for rigidity suggesting trauma or infection. Look for evidence of encephalopathy: hepatomegaly, ascites, caput medusae
7. Skin: Assess for rashes, petechiae, track marks. This can be a clue to infection, trauma, intoxication, or withdrawal.

8. Neurologic: A complete neurologic exam is often difficult to perform as it requires cooperation on the part of the patient. Assess for cranial nerves, strength, sensation, coordination, reflexes, and gait; focal deficits suggest a medical cause of psychosis.

## Emergency Diagnostic Tests and Interpretation

The lab and radiologic studies are dependent on the clinical presentation. Indicated studies may include electrolytes, acetaminophen levels, salicylate levels, LFTs, ammonia, PT/INR, thyroid studies, HIV, pregnancy test, ECG, CXR, UA, urine culture, blood culture, CT head, LP. Increasing age, preexisting medical comorbidities, the absence of past psychiatric history warrant, whereas young patients with a known psychiatric history, normal vitals/physical exam, a classic toxidrome or admitted drug use may not require lab or imaging studies unless the patient's mental status fails to improve on serial assessments. Evidence of trauma, the use of anticoagulants, or

a n abnormal neurologic exam should prompt a CT head to assess for a bleed or lesion. However, note that inpatient psychiatric facilities may require testing independent of clinical status.

## Emergency Treatment Options

These are divided into medical and psychiatric.

### Initial Stabilization

As always, ensure airway, breathing, and circulation. Ensure a safe and if possible, a low-stimulus environment to minimize agitation. This may be done by attempting to redirect the patient verbally and turning down the lights and decreasing the number of people in the room. Temporary physical restraints and emergency pharmacologic intervention may be needed if the patient escalates. Psychotic patients will benefit from an antipsychotic agent.

### Medications

Antipsychotics and benzodiazepines are the mainstays of treatment for acutely



psychotic patients who are agitated or violent. These may be given alone but are often given in conjunction and will be beneficial not only to the patient with psychosis secondary to primary psychiatric condition but also in various sympathomimetic toxidromes as well.

1. Benzodiazepines: For the acutely agitated patient, the benzodiazepines of choice are typically midazolam and lorazepam, given intravenously or intramuscularly. Midazolam may be preferred as it has a shorter time of onset as well as a shorter duration of action compared to lorazepam. Generally, diazepam is avoided due to its long half-life. In the elderly or pediatric patients, use lower doses. Watch for paradoxical disinhibition.

2. First-generation antipsychotics: haloperidol and Droperidol. Potential side effects include extrapyramidal side effects, e.g., dystonic reaction, as well as potential arrhythmias, especially long QT leading to torsades.

When possible, get a baseline ECG prior to administration.

3. Atypical Antipsychotics: Olanzapine, Ziprasidone, Quetiapine, Risperidone. These drugs also cause QT prolongation but generally have fewer extrapyramidal side effects.

## Disposition Decisions

Admission to the hospital: This is appropriate in patients with an identified underlying medical cause of psychosis or patient's in whom no underlying medical cause has been found, but a medical condition is suspected, for example, patients with no prior psychiatric history or patients with abnormal vitals. Inpatient admission may be necessary to follow up on studies, particularly cultures.

Discharge to home: This may be appropriate for patients with substance-induced psychosis who, either with the help of medications or simply the tincture

of time, have returned to their baseline mental status.

Admit to the psychiatric facility: This is appropriate for patients with higher symptom burden who have been medically cleared; dangerousness to self or others, or inability are unable to care for themselves due to psychosis warrant admission.

**References and Further Reading**, click [here](#)

# Stabilization and Management of the Acutely Agitated or Psychotic Patient

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by Michelle Chan, Nidal Moukaddam, Veronica Tucci

## Case Presentations

### Case 1

*It is a quiet Wednesday night in the emergency department when you suddenly hear someone coming down the hall continuously spouting out a string of profanities. You leave the comfort of your chair to see what the commotion is all about only to find a 37-year-old female brought in by police for altered mental status. She is acutely agitated on presentation, spouting obscenities non-stop, refusing to answer questions and uncooperative with a physical exam.*

### Case 2

*As you are pondering your next step, you see the paramedics wheeled an older gentleman past you and into the next room. You step into the next room to get a report. The family is at the bedside and states the patient is an 82-year-old male with a history of hypertension and BPH who has been increasingly*



Audio is available [here](#)

*confused and aggressive over the past two days. You note that he is mildly tachycardic when you hear the PA system announce, “Security is needed in the critical care hallway.”*

### **Case 3**

*A nurse pops her head into the room and requests your immediate assistance. You follow him down the hall and see your charge nurse along with three security officers trying to hold down a male patient. The patient, who appears to be in his late twenties, is actively kicking and trying to bite and spit at the medical staff. He appears flushed and diaphoretic.*

## **General Approach and Critical Bedside Actions**

### **General Approach and Key Concepts**

The first steps in evaluating any patient who presents to the emergency department are to assess and ensure that the patient’s airway, breathing, and circulation is intact. However, when presented with an acutely agitated or psychotic patient, even before assessing the ABC’s, you should ask yourself if the patient poses an immediate threat to the safety of both your patient or your medical staff.

If you determine that the patient poses an imminent threat to self or staff, a number of factors should be considered prior to administration of medications in order to achieve rapid stabilization without over-sedation or use of undue force. These factors include age, known psychiatric history, known or suspected substance abuse, and severity of agitation. Case 3, above, is a common scenario in the ED where a patient clearly poses an immediate threat. Clearly call out the medication(s) and dosage(s) you would like, and while it is being prepared, attempt de-escalation techniques. Appropriate pharmacologic and non-pharmacologic agents will be further discussed under Emergency Treatment Options.

Once immediate safety has been established, make sure the patient is placed on monitors and a full set of vitals, including temperature, is acquired. This should be followed by a thorough

primary survey. A commonly used mnemonic is “ABCDE,” where A = airway, B = breathing, C = circulation, D = disability, and E = exposure. Disability refers to assessing a patient’s level of consciousness and quickly screening for reversible causes of altered mental status. This includes examining pupils and checking blood glucose. Hypoxemia and hypotension should be noted in the vitals. Exposure is a critical bedside action that is easily neglected. All patients who present with acute agitation or psychosis should be fully exposed and changed into a hospital gown. This allows for rapid visual assessment for overt signs of trauma, as well as the opportunity to check for concealed weapons.

## Differential Diagnoses

The differential for patients presenting with acute agitation or psychosis is long (see the list for common causes), but is best divided into organic (primarily medical) and inorganic (primarily psychiatric) causes.

## Common Causes of Acute Agitation or Psychosis

### Medical

- Hypercalcemia
- Hypercapnia
- Hypoxia
- Infection (encephalitis, meningitis, sepsis)
- Substance-related (alcohol, hallucinogens, steroids, stimulants, synthetic marijuana)

### Psychiatric

- Bipolar disorder
- Posttraumatic stress disorder
- Psychotic depression
- Schizophrenia spectrum disorders

## History and Physical Examination Hints

History is often limited in patients presenting with acute agitation. Whenever possible, gather collateral information from family, emergency personnel and police. Key information to obtain include:

- Past medical history
- Past psychiatric history (include current psychotropic medications)
- Allergies
- Home medications
- Social history (alcohol use, substance abuse, living situation, caregivers, etc.)
- Recent health status (i.e., Has patient reported headaches in the past few days suggestive of a possible intracranial process or has an elderly patient reported dysuria suggestive of possible urosepsis)
- Baseline mental status

Although it is often said that history is 80% of the diagnosis, in cases of acute agitation, a thorough physical examination is all the more important due a limited HPI and review of systems. The physical exam of every acutely agitated patient should include a full neurologic exam and head-to-toe visualization for obvious signs of trauma or injury. A full neurologic exam may not always be possible during the initial assessment but should be completed as soon as feasible and prior to disposition.

When dealing with acute undifferentiated agitation due to a limited history and physical, a number of key signs and symptoms may help to at least narrow the differential to organic vs. inorganic causes. Organic causes of agitation tend to be associated with abnormal vital signs, disorientation, fluctuating symptoms, or signs of trauma, whereas, inorganic causes lack these features.

## Emergency Diagnostic Tests and Interpretation

Blood work, diagnostic tests, and imaging should be guided based on history and physical exam. Depending on your institution, laboratory tests required for medical clearance should also be taken into account. A basic metabolic panel and CBC, although not standard, is typically ordered for most patients with acute undifferentiated agitation or psychosis.

A urine pregnancy should be ordered on all women of childbearing age. When urine is unobtainable, consider a qualitative hCG or substituting blood for urine on a point-of-care pregnancy test.

In cases of suspected ingestion or substance abuse, consider checking acetaminophen and salicylate levels, as well as an EKG and measurable levels of prescription drugs to which the patient has access.

## Emergency Treatment Options

The goals for the treatment of acute agitation is early recognition, intervention before escalation into more violent behavior, and stabilization of life-threatening conditions. Therapy should be aimed at decreasing agitation and psychosis to the greatest extent possible without oversedation in order to allow for further assessment of the patient to determine the underlying cause. Interventions can be divided into nonpharmacologic and pharmacologic strategies.

Non-pharmacologic strategies include environmental interventions, de-escalation techniques, mechanical restraints, and seclusion. Environmental conditions are often difficult to control in the ED, but the concept is simple: create a safe space that minimizes stimulation. This means screening patients for weapons, removing objects that could be used as weapons (pens, chairs, or other loose objects), finding space away from



the noise and activity of the ED when possible, and dimming room lights. De-escalation techniques involve both verbal and non-verbal methods. Once again, the concepts are simple and should be applied as first-line techniques in the management of acutely agitated or psychotic patients. Successful use of de-escalation techniques will vary from situation to situation, but many times hinges on the ability to establish rapport with the patient quickly. Often times this can be accomplished by addressing a patient's basic needs of safety, hunger, and comfort. Provide reassurance that the patient is in a safe place, offer food, water or warm blankets, and make sure to address pain management. It is equally important to be mindful of personal space and avoid the threatening or confrontational behavior. Verbal de-escalation techniques involve maintaining a calm and respectful demeanor while acknowledging the patient's anger, frustration or agitation.

Although environmental and de-escalation strategies can be very effective, many times, acutely agitated or psychotic patients will require some form of pharmacologic intervention. The two major classes of drugs used for this purpose are antipsychotics and benzodiazepines. They may be used in combination or as monotherapy and are available in many formulations. In the case of acute agitation or psychosis, these medications are most often given parenterally, either intramuscularly or intravenously for rapid tranquilization. Refer to Table 2 for common agents and doses.

Choice of medication(s) varies greatly depending on personal preference; however, the two most commonly used agents are haloperidol and lorazepam. In patients with a known history or high suspicion for underlying psychosis, monotherapy with an antipsychotic such as haloperidol may be considered.

Whereas, in patients with undifferentiated agitation or psychosis, monotherapy with a benzodiazepine may be a better option due to its added anxiolytic effects and usefulness in cases of substance-related psychosis such as phencyclidine (PCP) use or alcohol withdrawal.

When the above interventions fail to stabilize an acute agitated or psychotic patient, physical restraints and/or seclusion may be necessary. It is important to understand, however, that these are methods of last resort and should never be used out of convenience or as a form of punishment. Seclusion differs from placing the patient in a safe and less stimulating environment in that seclusion involves involuntary confinement. Both seclusion and physical restraints are associated with increased morbidity and mortality.

## **Pediatric, Geriatric, Pregnant Patient and Other Considerations**

### **Geriatrics**

With age comes an increased risk for dementia which is a potential cause of acute agitation and psychosis in the geriatric population. Although effective in the management of psychosis, FDA issued a black box warning for olanzapine and ziprasidone regarding their use in elderly patients with dementia-related psychosis due to increased mortality in this population.

### **Disposition Decisions**

Once stabilization of a patient's acute agitation or psychosis is achieved, disposition should be guided by the underlying cause of agitation. Organic causes of agitation or psychosis such as sepsis, acute intracranial hemorrhage or severe metabolic disturbances warrant hospitalization until stabilization of the underlying cause is also achieved.

Whereas, patients who present with

substance-induced psychosis may be eligible for discharge if the patient is no longer clinically intoxicated and back to baseline mental status.

If after medical evaluation is complete, and no medical cause of agitation or psychosis can be determined, patients must then be evaluated for psychiatric causes. Admission criteria for acute psychosis due to an underlying psychiatric disorder primarily involves whether or not the patient is at high risk for continued harm to self or others. Admission is also strongly considered for first episodes of psychosis due to inorganic causes.

**References and Further Reading**, click [here](#)

# Medical Clearance – Suicidal thought/ideation

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by Veronica Tucci

## Case Presentation

*A 35-year-old female presents to the ED after the family called the paramedics for “bizarre behavior.” She notes that her family persuaded her to seek evaluation; however, they are not with her currently. She seems somewhat paranoid and tangential and is difficult to obtain a history from. On review of systems does endorse some mild abdominal pain and diarrhea. Her vital signs on arrival as recorded in triage are as follows: heart rate 135, blood pressure 110/90, respiratory rate 24, oxygen saturation of 96% on room air, temperature 100.7. When you speak with the family, they state that she has been agitated and paranoid. They are also concerned that she made suicidal threats while with friends.*

*On physical examination, you see an anxious appearing woman. She is tachycardic with an irregular pulse. Her extremities are dry with 1+ lower extremity edema. Her*



Audio is available [here](#)

*abdominal is soft and non-tender. She has no meningeal signs. She is tachypneic and has crackles in the lung bases. Her neurologic examination is non-focal. She reports suicidal ideation without a plan.*

## Introduction

Many medical conditions can present as psychiatric complaints. The case below will demonstrate the importance of the medical evaluation of these patients, as well as the need to keep a broad differential diagnosis. There are also medical problems which may exacerbate psychiatric symptoms or need to be addressed in order for a patient to be able to be transferred and safely managed at a psychiatric center. In addition to these scenarios, the patient's underlying psychiatric disorder may lead to an emergent medical condition, such as an overdose or a self-inflicted trauma.

Psychiatric complaints are common presentations for our ED (emergency department) patients and are ever increasing. Mental health-related visits increase from 1992 – 2001, most significantly in the areas of substance-related disorders, mood disorders, and anxiety. Pediatric mental health visits are also increasing. Interestingly, one study which showed this increasing trend found

that this did not hold true in the two areas that are mandatorily evaluated in the ED – suicidal ideation and acute psychosis. This suggested that the increase in visits was related to non-emergent psychiatric complaints that might be better managed by outpatient mental health professionals.

ED physicians are often tasked with evaluating these patients for medical problems prior to clearing them for possible psychiatric evaluation and admission. They must control the acute symptoms, attempt to determine the etiology of complaints (particularly functional vs. organic), provide appropriate initial treatment, and determine disposition. Of the utmost importance is identifying and treating immediate life-threatening problems. Historical data, mental status examination, physical examination, and appropriate ancillary testing are indicated.

This process has previously been termed “medical clearance.” While the

importance of this process cannot be stressed enough, is fraught with both intrinsic and extrinsic difficulties. Nevertheless, a thorough medical assessment is imperative to taking excellent care of this high-risk patient population.

One challenge is with the term “medical clearance” itself, which can be misleading. It means different things to different providers, and its overuse can result in poor patient care. No standard criteria exist for what medical clearance consists of, or even what the status of a medically cleared patient truly is. Complicating this further, different specialties have their own approaches to this evaluation. In addition, receiving psychiatric facilities often have their own requirements, irrespective of what the treating ED physician and psychiatrists believe to be medically indicated. Some have suggested modifying the term or replacing it instead with a thorough discharge summary. Another suggestion is to provide a summary of the evaluation

and treatment or using the term medically stable. There has also been shown to be wide variation in the comprehensiveness of medical clearance examinations.

## General Approach and Critical Bedside Actions

The most important first step in the assessment of this patient is to assess for abnormalities in the airway, breathing or circulation which may require immediate stabilization. A rapid blood glucose level should be obtained early on. The patient should be placed on a cardiac monitor and continuous pulse oximetry, and IV access should be established with tubes collected for blood work. Place the patient on oxygen by nasal cannula, and consider IV fluid.

With regards to the family’s concern about suicidal ideation, some precautions should be taken. This may include removing items and clothing from the room that could be used for self-harm. The patient should not be left alone and should have a staff member or a reliable

family member with them at all times. Importantly, she should not be allowed to leave the ED until the evaluation is complete.

## History Taking and Physical Examination Hints

For this patient, you will want to obtain further history including prior episodes, past medical history, associated complaints (cough, fever, heat intolerance, headache, neck pain/stiffness, changes in hair or skin, etc.), prior medications, drug and alcohol use, prior hospitalizations. It is important to obtain collateral for this patient who may be unable or unwilling to provide a full and accurate history for you.

A thorough history and physical are the starting point of any patient evaluation. Several studies looking at missed medical diagnoses in patients with psychiatric complaints have shown that these should have been identified if a proper history and physical were performed. Unfortunately, studies looking at the



thoroughness medical evaluations of these patients have often found them to be incomplete. An incomplete medical evaluation can lead to missed medical diagnosis, which can be dangerous for patients. One study found that “medically clear” had been documented in 80% of patients where a medical diagnosis should have been identified. One retrospective chart review found that complete vital signs were only documented 52% of the time.

All patients require a complete history, physical and mental status examination. This should be approached in an organized fashion in order to determine the etiology of their complaints as functional or organic. The medical evaluation of these patients should be no different than of those presenting with medical complaints. The history and physical should guide laboratory and other diagnostic testing and imaging. The information gathered from this will form the clinical picture.

History should be obtained from the patient in addition to those close to them like family and caregivers, and an effort should be made to confirm the information obtained from outside sources whenever possible. Sudden onset in changes in behavior, mood, or thought in a previously normal patient, or a deterioration in a patient with a chronic disorder should be suspicious for an underlying medical etiology.

Assessing for substance abuse, use, and changes are important. Also, inquire about adherence to their current medication regimen. Family and social stressor should be assessed. It is important to find out about medical comorbidities, or physical symptoms and complaints as these might also indicate a medical etiology (trauma, fever, etc.). Be aware that many medications can lead to changes in behavior, especially in at-risk groups. Physical complaints, abnormal exam findings, and abnormal vitals must be evaluated and addressed.

The mental status examination (MSE) plays a crucial role in the evaluation of these patients. The MSE needs to be focused and brief, and evaluate seven major areas (affect, attention, language, orientation, memory, visual-spatial ability, and conceptualization). Again, this should be structured and evaluate changes in alertness, cognition, behavior. Remember delirium, dementia, and psychiatric illness have significant differences in management and outcomes, and thus need to be identified. Delirium in the ED is associated with decreased survival. There are also alternatives to the traditional mental status examination. The quick confusion scale is a scoring system that was published and is quickly obtained, easily calculated, readily interpreted score.

## Differential Diagnosis

In the above patient, the following is a list of possible etiologies for her symptom: sepsis, diabetic ketoacidosis, pneumonia, pulmonary embolism, meningitis, encephalitis, hyperthyroidism/thyroid

storm, schizophrenia, bipolar disorder, psychosis, salicylate ingestion, acute intoxication, alcohol withdrawal/delirium tremens. There are several abnormalities in history and physical examination which suggest that the patient's symptoms are not primarily psychiatric in origin.

Alterations in mental status may incorrectly be attributed to psychiatric diagnoses. A review looked at 64 cases of patients admitted to the psychiatric ward, whom were later found to actually have a medical diagnoses that explained their symptoms. The etiologies identified included intoxication, withdrawal syndromes, overdose. In this, they noted that none had an appropriate medical screening examination performed. In another study looking at factors which may have contributed to a patients symptoms being attributed to a psychiatric problems instead of a medical one, found that these patients had a lower rate of complete history, physical examination, cognitive assessment, indicated ancillary testing and treatment

of abnormal vital signs in comparison to patients admitted to medical units.

Some special groups are at increased risk of having a medical etiology of their complaints, and care should be taken when evaluating these patients. Several prior studies have identified these as the elderly, those with substance abuse, those without a prior psychiatric history, and those with pre-existing or new medical complaints. Intoxicated patients represent a particular challenge. In addition to often providing a limited history, they may express certain complaints (like thoughts of self-harm), only while intoxicated. A study looked at 100 consecutive alert patients with new psychiatric complaints. They excluded those obviously intoxicated, prior diagnosis of abnormal behavior, those with medical complaints and overdose or suicide patients. For all patients, they then performed a history, physical, panel of laboratory tests, CT scan of the head and lumbar puncture if febrile. They concluded that 63/100 patients had an

organic etiology of their symptoms. A study looking at 658 psychiatric outpatients receiving medical and biochemical evaluation found the incidence of medical disorders producing psychiatric symptoms at 9.1%. The etiologies included infectious, pulmonary, thyroid, diabetic, hematopoietic, hepatic and CNS. Another study of 100 psychiatric patients who had been previously medically screened found that 46% had a medical illness that caused or exacerbated their symptoms and 80% of these required treatment. They concluded that a battery of laboratory and ancillary testing would have identified the majority of these.

## **Emergency Diagnostic Tests and Interpretation**

The following diagnostic testing should be considered in the above case.

**Table 11.1** Medical Clearance - Suicidal Thought/Ideation Diagnostic Tests

TEST	COMMENT
Complete blood count	Anemia, hematologic abnormality
Complete metabolic panel	Metabolic abnormality, uremia, liver failure, renal failure
Electrocardiogram	Arrhythmia, evaluation of tachycardia and irregular pulse
Chest X-ray	Pneumonia, heart failure, other etiology of tachypnea
Urinalysis/Urine culture	Source of sepsis
Blood cultures	Sepsis evaluation
Troponin and BNP	Heart failure
Thyroid function studies	Hyperthyroidism / thyroid storm
CT scan of the brain	Abscess, meningitis, mass
Lumbar puncture	Meningitis/encephalitis
Alcohol level/urine drug screen	Intoxication, may be required at psychiatric facility
Acetaminophen and salicylate levels	Commonly ingested in suicide attempts

Original by author

While everyone can agree that these patients deserve a complete history and physical, the role of laboratory and ancillary testing is less well delineated and is often viewed differently among ED and psychiatric physicians. What studies are required for medical clearance of the psychiatric patient, and whether this process should be standardized, or be performed on a case by case basis, is the source of much controversy.

The American College of Emergency Physicians published a clinical guideline on the subject. They suggest that diagnostic evaluation should be directed by the history and physical and routine laboratory testing of all patients is of very low yield. Routine urine toxicology screens in awake, alert, cooperative patients do not affect ED management, and using this screening in the ED because of the requirement of receiving psychiatric facilities or service should not delay evaluation or transfer. They also say that patients' cognitive abilities rather than a specific blood alcohol level should

be the basis of beginning the psychiatric assessment and recommend considering a period of observation to determine if symptoms resolve as intoxication resolves. A study looked at patients with isolated psychiatric complaints and past medical history of psychiatric disorder. None of these had positive screening laboratory or radiograph results. The remaining patients had a presenting medical complaint as well, and these complaints directly correlated with the need for labs and radiography. They concluded that patients with a psychiatric complaint with a documented past psychiatric history, negative physical findings and normal vital signs, who deny current medical problems did not require further labs or testing in the ED.

Another systematic review of the literature indicated that history, physical examination, review of systems, and tests for orientation had relatively high yield for detecting active medical problems. Routine laboratory testing was relatively low yield. However, four groups were at

serious risk for medical problems, and these included the elderly, substance users, patients with no psychiatric history, and patients with pre-existing medical disorders and/or concurrent medical complaints. A retrospective chart review of pediatric patients presenting to an academic pediatric emergency department for medical clearance for an acute psychiatric emergency found that screening laboratory tests resulted in few management or disposition changes in patients if they had a noncontributory history and physical, but did result in an increased length of stay. The costs of routine testing given the low yield also should be considered.

## Emergency Treatment Options

The patient presenting above has several abnormal findings on history and physical which make a medical etiology more likely. While safety precautions for self-harm and levels of commonly ingested medications should be obtained, the

more pressing concern is stabilizing this patient from a medical standpoint.

During her stay, she becomes more tachypneic and supplemental oxygen is placed. Her EKG reveals atrial fibrillation with a rapid ventricular response. Her troponin and BNP are mildly elevated. Her CXR reveals signs of heart failure and pulmonary edema. Her TSH  $< 0.01$  and her T4 level is  $> 100$ . Overall, this is suggestive of hyperthyroidism/thyroid storm as the etiology of her psychiatric complaints.

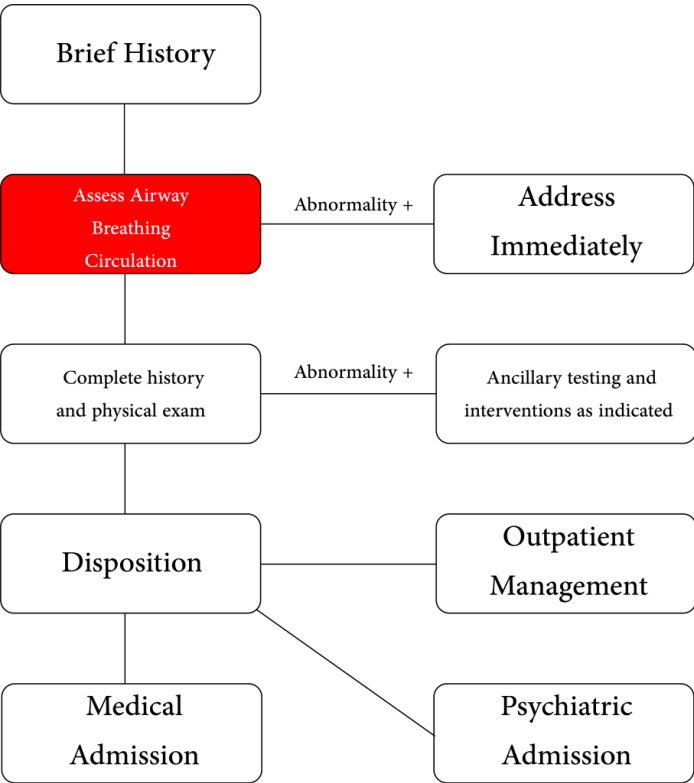
Propanolol is promptly administered in a dose of 1 mg IV over 10 minutes. Propylthiouracil (PTU) is then administered in a dose of 200 mg and iodine is administered 1 hour later. Steroids are given to prevent the conversion of T3 to T4. She is admitted to the intensive care unit for thyroid storm. She has a prolonged and complicated hospital course and is discharged several weeks later. Her psychiatric symptoms resolved with her medical treatment.

In this case, the appropriate disposition of the patient is the intensive care unit. In general, In patients presenting with psychiatric complaints, the medical screening exam should identify medical problems which require admission, or concurrent medical issues which may inhibit their ability to go to a psychiatric facility. However, if after the evaluation none of these medical issues are identified, or they are adequately treated in the ED, the patient needs to be evaluated for the need for psychiatric admission. In a patient with suicidal ideation, there are several components to be considered.

Severe anxiety, panic attacks, a depressed mood, a diagnosis of major affective disorder, recent loss of interpersonal relationship, recent alcohol or drug abuse coupled with feelings of hopelessness, helplessness, worthlessness, global or partial insomnia, anhedonia, inability to maintain a job and recent onset of impulsive behavior are predictors of suicidal behavior.



Diagram 11.1



Original by author

There are several clinical rating scales in suicide risk assessment. An example of one is the SAD PERSONS scale. It stands for: Sex, Age, Depression, Previous attempt, ethanol abuse, rational thinking loss, social support lacking, organized plan, no spouse, sickness. One point is given for each.

Assessing the risk of suicide is complicated, and complete psychiatric

evaluation is ultimately needed. A study looking at several risk assessment scales found that in general, they overestimated suicide risk. They did note that they might help highlight important concepts and risk factors. This may be particularly useful for non-psychiatric medical personnel or junior residents. They may help identify high-risk patients in the ED early in assessment, and those that may need psychiatric referral.

Conclusion

The role of the ED provider in psychiatric care is increasing, and external resources are often inadequate. A study of California EDs showed that there are limited mental health resources for suicidal patients. It suggested the need for more regional solutions including improved access to mental health personnel and follow of suicidal patients and community mental health resources for patient referrals. The same is true in the pediatric patient. The ED physician plays an important role in the pediatric ED in the stabilization and management of a

mental health crisis, the discovery of mental health issues in ED patients, and approaches to advocating for improved recognition and treatment in mental illness in children. The ED evaluation of pediatric mental health is crucial to the child’s long-term care and treatment.

It is important to note the overall significance of medical problems in the population of patients with psychiatric disorders, and the challenges that they face interfacing with and accessing the medical community. A study of this population out of Nova Scotia showed increased mortality from cancer, which may be attributed to delays in detection or initial presentation and difficulties in communication and access to healthcare contribute to this finding. Another study looked at compulsory community treatment in this patient population. They saw a reduction in all-cause mortality in their intervention, group which they stated that might be partially explained by increased contact with health services in the community. Looking at 200 patients



receiving psychiatric care in the outpatient setting for schizophrenia and affective disorder diagnosis, both groups had greater odds of having comorbid medical conditions than those in the general population.

In addition to the challenges of assessment, these patients present logistical difficulties in the ED setting. Patients with psychiatric related complaints have long lengths of stays in the ED. Older individuals, the need for hospitalization, restraint use and diagnostic testing prolonged the length of stay. Drug and alcohol screening also led to delays. They also tend to have high rates of readmission. Predictors of 12-month readmission and ED revisits for patients with substance abuse, and mental health-related complaints were highest in those with dementia, psychotic disorders, autism, impulse control disorders and personality disorders.

The care of patients with psychiatric complaints is complicated and

challenging. The ED physician is tasked with the initial assessment both of psychiatric risk and medical clearance. Care should be taken to stabilize any life-threatening condition and then to try to differentiate a functional versus organic cause of the patient's symptoms. They must also assess for any underlying medical problems that may exacerbate the patient's symptoms or need to be managed at a psychiatric facility. The psychiatric disorder itself may also lead to a life-threatening medical condition that needs to be threatened or treated. This is a very important part of the care of this challenging patient population.

**References and Further Reading**, click [here](#)

## Chapter 12

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# Selected Orthopaedic Problems



# Back Pain

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by Funda Karbek Akarca

## Case Presentation

*A 45-year-old age male presented to the emergency department with severe back pain after lifting a heavy object. He described the pain radiated to the right leg. He had difficulty with walking. His medical history revealed no additional diseases except for occasional back pain. The vital signs were normal. The physical examination showed palpable peripheral pulses, no motor or sensory deficit, no drop foot or murmur in the abdomen. Straight leg raising test is positive at 45 degrees. Palpation of the vertebrae revealed no tenderness on spinous processes but paravertebral muscles spasm. The patient's pain decreased after resting in the supine position, muscle relaxants, and analgesics. The patient was discharged with a recommendation of neurosurgery visit in ten days.*

## Critical Bedside Actions and General Approach

Back pain is a common problem and affects up to 90% of the general population at some point in their lives. It is the fifth leading cause and accounts for 2% to 3% of emergency department visits. Although most back pain is due to a benign and self-limiting reason, a minority of patients may face a risk of permanent neurological damage or death.

Acute, non-traumatic low back pain can be divided into three groups: musculoskeletal causes with no neurologic deficits, musculoskeletal causes with neurologic deficits and other causes that can present with back pain.

- 👤 Check vital signs; especially fever
- 👤 Learn the history of current illness; pain duration, how the pain started and spread.
- 👤 Take medical history; disc herniation history, recent spinal anesthesia or surgery,

corticosteroid or anticoagulant use, cancer

- 👤 Make an orderly and thorough physical examination
- 👤 Order necessary imaging and labs
- 👤 Assess the risks and consider the potentially life-threatening or debilitating diagnoses.

• Abdominal aortic aneurysm

- Aortic dissection
- Upper Urinary Tract Infection, renal infarction, renal colic
- Abdominal infection (cholecystitis, cholangitis, pancreatitis, retroperitoneal abscess)
- Abdominal neoplasm

## Differential Diagnoses

### Spinal origin

- Musculoligamentous
- Discopathy
- Fracture
- Spondylolisthesis
- Vertebral osteomyelitis
- Spinal epidural abscess
- Spinal epidural hematoma
- Neoplasm/metastatic disease

### Nonspinal causes

## History and Physical Examination Hints

In many patients, a thorough history and physical examination are essential and sufficient for diagnosis. The tips indicating severe pathologies should be investigated (red flags are shown in Table 1). Additional questions are whether the patient has a similar pain before, has any prior diagnosis related to this complaint or receive any treatment.



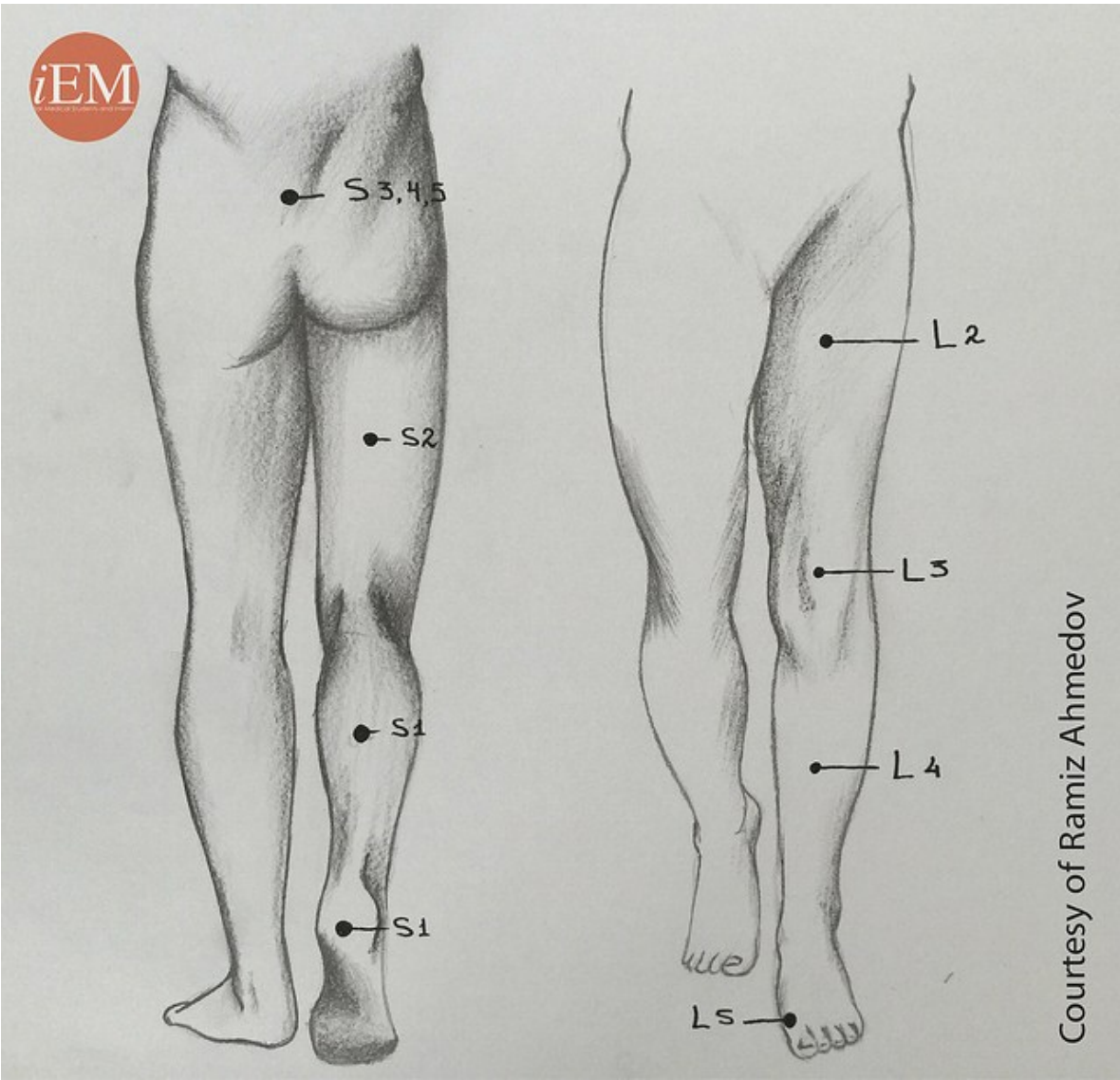
**Table 12.1** Red Flags In Back Pain

HISTORY	PHYSICAL EXAMINATION
Pain duration more than 6 weeks	Fever
Age;child or elderly	Major motor weakness esp. bilaterally
Fever, malaise, weight loss	Saddle anesthesia
IV drug use	Perianal sensory loss
Corticostreoid use	Urinary retansion
Cancer history (especially bone metastasis)	Anal sphincter laxity
Trauma esp. in elderly	Fecal incontinance
Recent instrumentation or spinal anesthesia	

*provided by author*

The typical back pain from muscles or ligaments is generally easily localizable, increases with movement and decreases after rest. The pain rarely radiates, but when it does, it radiates to the pelvis. It the pain radiates below the knee, it may point to L3 nerve root radiculopathy. However, about %90 of the disc herniations relate to L4-5, L5-S1 regions (Illustration 12.1). The location of pain and nerve root innervation).

**Illustration 12.1**



The duration of pain is important. The musculoskeletal pain generally limits itself in 4-6 weeks. Consider malignancy if the pain lasts longer.

The patients under 18 and over 50 years old are under risk for non-musculoskeletal pathologies.



The systemic symptoms are another tip for non-musculoskeletal pain. Fever, tremor, night sweating, anorexia, unexplained weight loss are significant for infection and malignancy. Immunocompromised patients (diabetic, the corticosteroid use, IV drug use) may not develop a healthy inflammatory response and accordingly the systemic symptoms; therefore, they may require further investigations. Consider spine infections in IV drug users.

The prior cancer diagnosis should make the physician consider spine metastases. Especially breast, lung, thyroid, kidney, prostate cancers and lymphoma tend to metastasize to the spine.

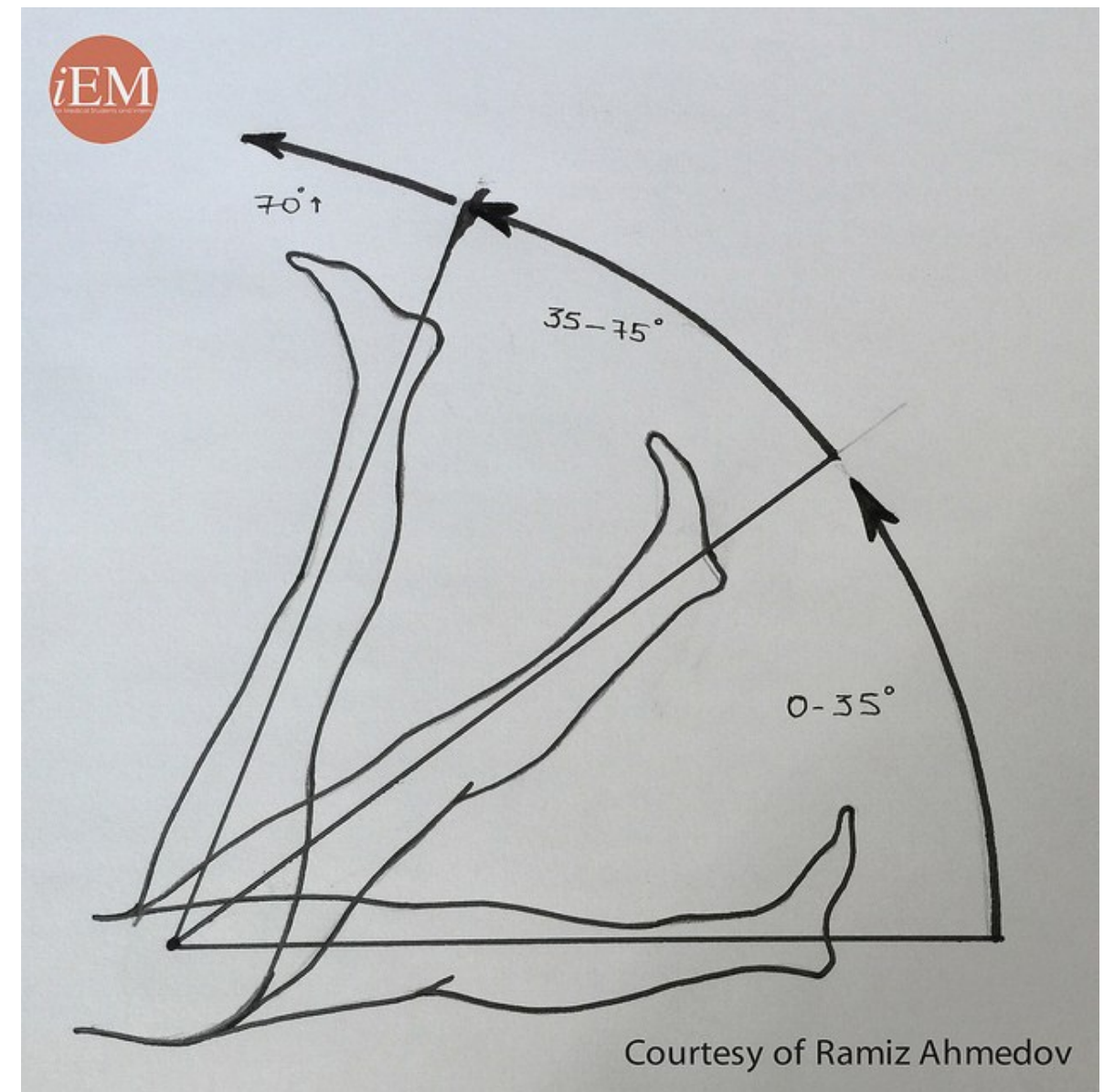
The physical examination aims to detect the risk factors and neurologic deficits. Check the vital symptoms at the beginning. Fever may point to spinal infections, but its sensitivity is low.

Lying flat will decrease the musculoskeletal pain. If the pain increases with lying, consider nephrolithiasis, spine infection, abdominal aortic aneurysm. Check all patients for abdominal tenderness, a palpable pulsatile mass, and murmur.

Assess vertebral tenderness; erythema, increased heat, purulent lesion over the adjacent skin. Evaluate each vertebral spinous processes individually.

Apply straight leg raise test (Lasegue's test) by elevating each leg slowly while the patient in supine position. Increased pain or reproduced sciatic symptoms during test means positive (Illustration 12.2). Straight leg raise test).

**Illustration 12.2**



If the pain occurs at 30-35 degree angle, it is considered significantly positive. The radicular pain worsens with ankle dorsiflexion and improves with plantar flexion. Straight leg raise test highly sensitive but not specific. Contralateral (opposite or well-leg) straight leg raise is highly specific but poorly sensitive for

L4-5 or L5-S1 radiculopathy. In other words, a negative contralateral straight leg raise is useful to exclude disk protrusion (see videos [1](#) and [2](#)).

Evaluate ankle and the first toe's dorsiflexion and plantar flexion for L5-S1 nerve roots. Examine patella and ankle deep tendon reflexes. Evaluate bilateral dermatomes and check for saddle anesthesia. Test the sensation of light touch along dermatomes from L1 to S1. Standard dermatomal charts can be helpful, but there is variability between individuals, and this test is highly subjective. In the upper lumbar roots, there is often a significant overlap. The L4, L5 and S1 nerve roots are the most discrete levels for testing. Additionally, these are the most often affected lumbar discs.

The rectal examination is not routinely indicated. However, in case of bladder or bowel incontinence, it is mandatory. Decreased rectal tone and the sensorial defect may make the physician consider epidural compression syndrome.

## Emergency Diagnostic Tests and Interpretation

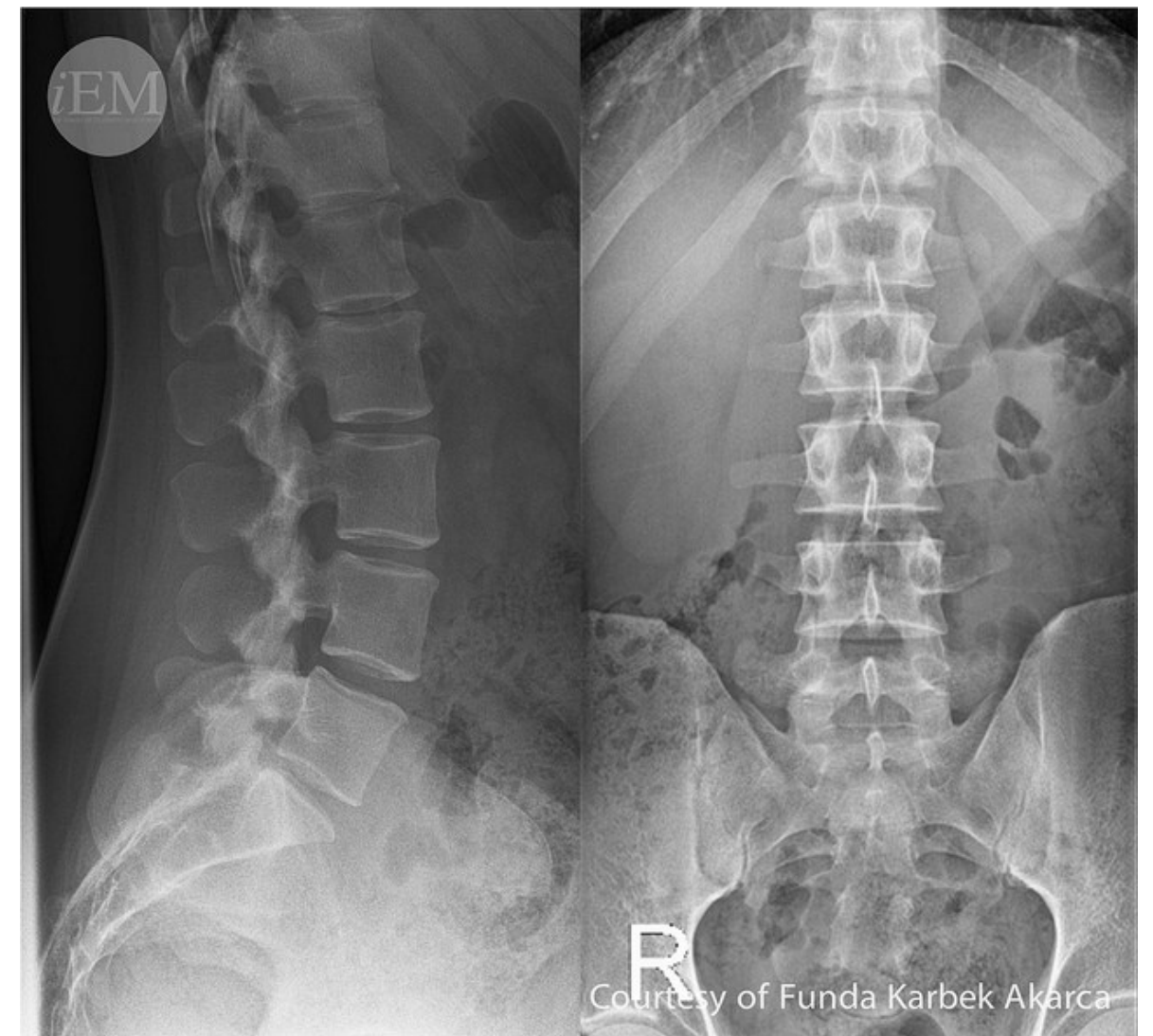
History and physical examination are essential. Laboratory testing is generally not useful.

Elevated white blood cell (WBC) may point to the infectious diseases. However, WBC is high only in the two-thirds of the patients with a spinal epidural abscess.

Inflammatory markers (Eritrosit sedimentation rate or C-reactive protein) are highly sensitive but not specific for epidural abscess or cancer.

Lumbosacral anterior-posterior and lateral X-rays are indicated in case of suspected fractures, especially in patients over 50 years old.

**Image 12.1** Normal lumbosacral X-ray. Lateral (left), AP (right)





**Image 12.2** Lumbar flattening



**Image 12.3** Normal spinal CT lateral view



The computed tomography can be useful in fractures or facet joint pathologies.

**Image 12.4** L2 compression fracture



Bedside ultrasound should be performed if the patient has urinary retention or suspected abdominal aortic aneurysm dissection.

Magnetic Resonance Imaging (MRI) visualizes abscess, metastatic lesions, and hematoma. Additionally, patients with neurological deficits requiring urgent surgery may necessitate MRI.

**Image 12.5** Discopathy



**Image 12.6** L2 compression fracture



**Image 12.7** Pneumonia secondary to spinal abscess



## Emergency Treatment Options

### Initial Stabilization

Structured management is essential in the emergency department. Stabilization is a priority. A critical abnormality in the vital signs and clinical may lead to the



early intervention in the life-threatening diseases and permanent neurological damage is at stake. After stabilization, pain control should be provided.

## Medications

- Pain is the main symptom. Non-steroidal anti-inflammatory drugs (NSAIDs) are considered as first-line therapy for acute back pain. Ibuprofen has less adverse effects and toxicity.
- Acetaminophen may be another choice.
- Opioids analgesics should not be administered more 1-2 weeks.
- The muscle relaxants are another treatment choice.
- The use of steroids is not recommended due to lack of evidence.
- The patients should return to their daily activities after a few days of bed rest.

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

### Pediatric considerations

In the pediatric age group, back pain is unusual. Consider the infectious causes. Ibuprofen is the preferred analgesic in this population.

For children: Ibuprofen; Infants and Children <50 kg: Limited data available in infants <6 months: 4 to 10 mg/kg/dose every 6 to 8 hours; maximum single dose: 400 mg; maximum daily dose: 40 mg/kg/day. Children ≥12 years: Refer to adult dosing.

### Elderly considerations

Consider fractures in elderly patients with relatively minimal trauma. Additionally, consider non-musculoskeletal causes of back pain, such as abdominal pathologies, aortic aneurysm or dissection.

### Pregnant considerations

Back pain is frequent in later pregnancy. The neurological deficit is infrequent. Pain control via analgesics and back strengthening exercises are recommended. Paracetamol is considered safe in pregnancy and should remain the first-line treatment for pain and fever. General Dosing Guidelines: 325 to 650 mg every 4 to 6 hours or 1000 mg every 6 to 8 hours.

## Disposition Decisions

### Admission Criteria

- Patients with uncontrolled pains
- Patients with progressive neurological deficit
- Patients with symptoms of cauda equina syndrome
- Patients with infectious, vascular or malignant pathologies



## Discharge Criteria

Patients with musculoskeletal pain without neurological deficits may be discharged after pain control

## Referral

Patients should be referred to neurosurgical or orthopedic surgery departments.

## Pearls and Pitfalls

- Musculoskeletal causes with no neurologic deficits include degenerative spine disease, muscular or ligamentous injury and mostly acute disc pathologies. These patients have normal neurologic examination but have severe pain.
- If the patient has positive neurologic findings during the examination, consider sciatica with radiculopathy and the severe other diagnoses.
- Don't forget that new neurologic physical findings suggest severe disease and serious diseases have normal neurologic examination findings. Emergency physicians must think broadly and consider nonspinal causes of back pain like an aortic aneurysm.

**References and Further Reading**, click [here](#)

# Lower Extremity Injuries

by Ayse Ece Akceylan

## Hip

### Case Presentation

*A 75-year-old male with a history of osteoporosis presented to the emergency department after falling on his right side. He complained of pain on the right hip. His vital signs were normal. His right leg was in abduction and external rotation and shorter than the left leg. Distal pulses were palpable. An anteroposterior (AP)*

*pelvis x-ray showed a femoral neck fracture. The patient was admitted to the orthopedics ward for surgical repair.*

### Critical Bedside Actions and General Approach

Preserving function, preventing infection and assuring perfusion of the limb should be the goals. Proper diagnosis and treatment are essential for establishing these goals.

- Check vital signs
- Learn mechanism of injury
- Take medical history

- Make an orderly and thorough examination
- Order necessary imaging and labs
- Noncritical orthopedic injuries should be treated only after more threatening injuries have been addressed.

## Differential Diagnosis

The patient might have one or more of the following:

- Hip fracture
- Hip dislocation
- Acetabular fracture
- Neurovascular injury

## History and Physical Examination Hints

- Note systemic illnesses, known metabolic disorders and medications. These may provide clues that lead to uncovering the reason behind what may seem like a simple trauma. (I.e., a fall may be the result of a cardiovascular event.)

- Visual inspection and palpation: look for tenderness, pallor, ecchymosis, deformity, abrasions, lacerations, and open wounds. An open fracture is a fracture associated with overlying soft tissue injury, creating communication between the fracture site and the skin. Even a puncture wound extending to the depth of an underlying fracture is considered an open fracture. Open fractures are usually classified by their severity, based on the size of the overlying laceration, the extent of tissue damage, lack of bone coverage, the kinetic energy of the injuring force, and evidence or likelihood of significant contamination.
- In a femoral fracture, the limb is shortened and externally rotated.
- Most hip dislocations are posterior. In posterior dislocations, the limb is adducted, internally rotated, and shortened.

- In anterior dislocations, the limb is abducted, externally rotated, and shortened.
- Check neurovascular status: Femoral nerve and artery may be injured with anterior hip dislocations. The sciatic nerve may be injured with a posterior hip dislocation or a hip fracture. Check pinprick sensation, light touch and motor function. Also, check femoral, popliteal, dorsalis pedis, and posterior tibial pulses.
- Dislocations and fracture-dislocations of the hip are two true orthopedic emergencies. The hip joint possesses impressive inherent strength and stability; therefore, considerable force is required to produce these injuries. It is highly recommended that in the presence of this type of injury, patients be managed as major trauma victims.

## Emergency Diagnostic Tests and Interpretation

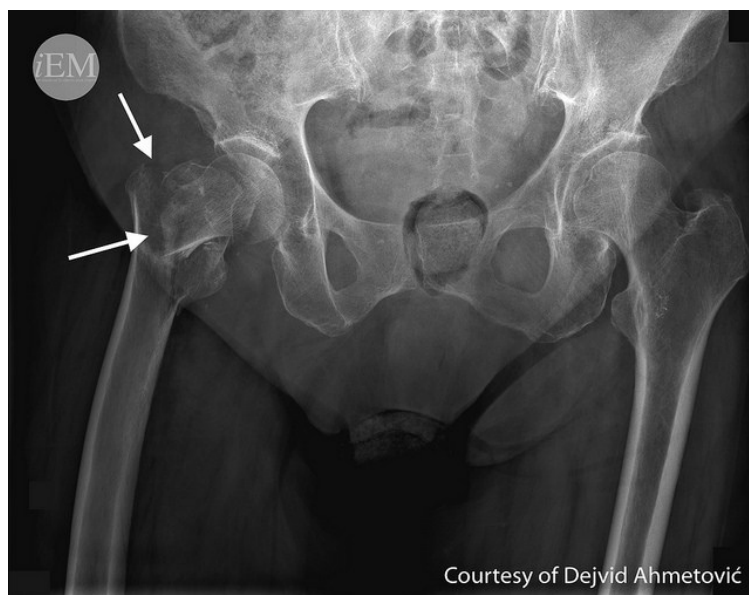
- Anteroposterior (AP) and lateral radiographs of the hip are usually

sufficient to diagnose hip dislocations and fractures. (See Image 12.8 and 12.9)

**Image 12.8** Right hip dislocation



**Image 12.9** Fracture of the femoral neck and peritrochanteric fracture



- Significant pain with weight bearing in the face of normal radiographs should raise suspicion for occult fracture, especially at the femoral neck or acetabulum.
- If there is a suspicion of fracture but plain radiographs appear negative, computed tomography (CT, See Image 12.10) or magnetic resonance imaging (MRI) may be used for diagnosis.

**Image 12.10** Left acetabular fracture



- If there is suspicion of vascular injury, cardiovascular surgery consultation and Doppler flow ultrasound, plain

angiography or CT Angiography are necessary.

## Emergency Treatment Options

- Most femoral and hip fractures need operative repair. Consult an orthopedic surgeon. Meanwhile, immobilize the extremity to prevent it from further damage.
- If a fracture is suggested clinically but radiographic films appear negative, the patient should initially be treated with immobilization as though a fracture were present.
- Patients with a traumatic fracture of the hip or femur may lose about 2 to 3 units of blood at the fracture site and require blood transfusions. Therefore, order blood type and crossmatch for at least 2 units of blood.
- Dislocated hips need to be reduced as soon as possible, under procedural sedation and analgesia (see videos 1 and 2).

- The sooner a joint is relocated, the better, to avoid neurovascular compromise. Also, delays cause swelling and muscle spasm, which hinder reduction. Use adequate analgesia or conscious sedation before attempting relocation. The emergency physician sometimes may be unable to reduce a dislocation. Orthopedic consultation is necessary in such cases.
- For hip dislocations, after reduction, the legs are immobilized in slight abduction with a pillow between the knees, and the patient should be sent for radiographs. Check neurovascular status before and after all reductions and after administration of immobilization.
- Withholding Oral Intake: Any patient who might go under general anesthesia or procedural sedation should not be allowed to eat or drink from the moment of arrival until the need for, and timing of, such a procedure has been ascertained.

- In case of open dislocation/fracture, remove gross contaminants from the wound and irrigate the injury thoroughly. Apply saline-soaked sterile gauze, and splint the injured leg. If a significant deformity is present, immediate reduction before splinting is indicated. Administer tetanus immunoprophylaxis as appropriate (Tetanus booster: 0.5 ml (Tdap) IM, Tetanus immunoglobulin: 250 IU IM if not previously immunized against tetanus). Start the patient on intravenous antibiotics. For injuries with mild to moderate contamination, a first-generation cephalosporin such as cefazolin 1–2 g (pediatric dose: 20 mg/kg IM/IV) is usually sufficient.<sup>2</sup> Heavily contaminated wounds require the addition of gram-negative bacterial coverage, typically an aminoglycoside such as gentamicin 1.5–2 mg/kg IV (pediatric dose: 2–2.5 mg/kg IV). Adding either penicillin G 4–5 million U IV (pediatric dose: 50,000 U/kg IV) or, if penicillin allergic, clindamycin or metronidazole as a third antibiotic is necessary for farm- or soil-related crush

injuries, in which contamination with *Clostridium perfringens* can be present. Early surgical intervention for debridement and irrigation is crucial, so emergency orthopedic consultation is indicated. Administer analgesics as necessary (**Morphine** sulfate: 2–10 mg (pediatric dose: 0.05–0.1 mg/kg per dose IV or equivalent analgesic)).

- In case of neurovascular injury, surgical consultation is necessary.

## Special Populations

- Treatment options are mostly the same for children, elderly and pregnant patients.
- In a fall, elderly patients may have sustained additional injuries; most commonly, these injuries involve a fracture of a vertebral body or wrist. Cervical spine and intracranial injuries also are considered.
- The dislocation reduction methods for patients with hip arthroplasty are the same as with a native hip.



- Fractures involving the physis, the cartilaginous epiphyseal plate near the ends of the long bones of growing children, are called Salter fractures. Damage to the epiphyseal plate during a child's growth may result in an aborted or deformed growth of the limb.
- Children who have sustained trauma at or near a joint may need comparison studies of the opposite extremity to differentiate fracture lines from normal epiphyseal plates or ossifying growth centers.
- The elderly are more prone to serious injury from low-energy mechanisms. The elderly are more susceptible to adverse outcomes following trauma because of comorbid diseases and physiologic changes that arise with aging.

## Disposition Decisions (admission, discharge, referral)

- Hip dislocations that cannot be reduced in the emergency department need to

be reduced in the operation room under general anesthesia.

- Hip fractures and hip dislocations (even if reduced in the ED) need to be admitted to the orthopedics ward.

# Knee

## Case Presentation

*A 60-year-old female presented to the emergency department with pain and swelling on her right knee after a fall. Her vital signs were normal, and she did not hit any other part of her body. Upon physical examination, there was tenderness and deformity on the right knee. The neurovascular examination was*

*normal. The x-ray revealed a comminuted patellar fracture (Image 12.11). The patient was admitted to the orthopedics ward for surgical repair.*

Image 12.11



Courtesy of AA Cevik

## Critical Bedside Actions and General Approach

These steps are the same as those mentioned above in the topic “hip.”

## Differential Diagnosis

The patient might have one or more of the following.

- Distal femoral, proximal tibial, proximal fibular fracture
- Knee dislocation
- Ligamentous injury
- Meniscal injury
- Popliteal artery injury
- Peroneal, tibial (less common) nerve injury

## History and Physical Examination Hints

- Note systemic illnesses, known metabolic disorders and medications, as mentioned above in the topic “hip.”

- Visual inspection and palpation – mentioned above in the topic “hip.”
- See Image 12.12 for open knee dislocation.

**Image 12.12**



- Check peripheral nerves: knee trauma, especially knee dislocations may cause peroneal nerve injury. Examine the peroneal nerve by testing the sensation

of the dorsum of the foot and by dorsiflexion of the ankle. The posterior tibial nerve may also be injured. This manifests with diminished plantar sensation and plantar flexion of the foot.

- Check vascular status: knee trauma may cause vascular injury. Check the popliteal, dorsal pedal and posterior tibial arteries.
- Palpate the extensor mechanism for tenderness and crepitation: quadriceps tendon, patella, patellar tendon, and tibial tubercle.
- Palpate the joint line (for meniscal or collateral ligament injuries)
- Check the range of motion of the knee
- Perform knee stability testing and meniscal testing (see videos 3 and 4)
- Comparison with the uninjured knee is helpful, especially for assessment of ligamentous laxity.

A grossly unstable knee after a traumatic injury should be assumed to be a reduced dislocation until proven otherwise.

(For more information, videos 5 and 6 will be helpful)

## Emergency Diagnostic Tests and Interpretation

The **Ottawa Knee Rule** and the **Pittsburgh Knee Rule** are useful for deciding when to order plain radiographs. Both criteria are sensitive for fractures, but the Pittsburgh criteria are more specific and can be applied to both children and adults. This approach is associated with an approximately 1% chance of a missed fracture. Therefore patients should be reevaluated in the event of persistent or progressive symptoms.

Ottawa Knee Rule [video](#)

If plain radiographs are indicated, obtain a minimum of an antero-posterior (AP) and a lateral view. Remember to examine

the joint above and the joint below the injury, not to miss associated injuries.

The joints above and below a fracture should generally be imaged for coexisting fractures.

Pre-and-post-reduction radiographs are advisable both before and after reduction of dislocations and fractures.

See Image 12.13 for comminuted tibial plate fracture involving the knee joint.

**Image 12.13**



In acute knee trauma, the goal of radiography is to rule out fracture. Because radiographs are not 100% sensitive, knee immobilization and orthopedic referral for reevaluation are options. When suspicion for a fracture is extremely high, CT or MRI can be used.

If there is suspicion of vascular injury, the same rules apply as mentioned above in the topic “hip.”

## Emergency Treatment Options

Most fractures concerning the knee joint need operative repair. Consult an orthopedic surgeon. Meanwhile, immobilize the extremity to prevent further damage.

If a fracture is suggested clinically, but radiographic films appear negative, immobilize the limb as though a fracture is present and consult an orthopedic surgeon.

**Patellar Fractures:** Nondisplaced fractures usually heal with a long leg cast



for 4 to 6 weeks. Displaced fractures are treated surgically.

**Patellar Dislocation:** After reduction, immobilize the knee in full extension for 3 to 6 weeks. Ice, elevation, non-weight bearing, and analgesia are beneficial in the acute setting. The patient can be discharged with a referral for a follow-up within 2 weeks. Watch this [video](#).

**Knee Dislocation:** To avoid tissue damage, the reduction should be attempted as soon as possible.. After reduction, immobilize the knee and call for an orthopedic consultation. Watch this [video](#).

For open fractures/dislocations, the same rules apply as mentioned above in the topic “hip.”

**Meniscal Injuries:** Unless the knee is locked and cannot be extended or flexed (which requires orthopedic consultation), a patient with a meniscal tear should be managed with analgesics, immobilization,

ice, non-weight-bearing status, and referral for an orthopedic follow-up.

In case of neurovascular injury, urgent surgical consultation is necessary.

**Controlling Pain and Swelling:** The early application of cold and elevation are effective in minimizing swelling or at least deterring its progression. Administer analgesics as necessary.

**Withholding Oral Intake:** same as mentioned above in the topic “hip.”

## Special Populations

Treatment options are mostly the same for children, elderly and pregnant patients.

## Disposition Decisions

- All dislocations (even if reduced in the ED) and most fractures need to be admitted to the orthopedics ward.
- Ligamentous and meniscal injuries can be sent home, with the advice of immobilization, elevation, ice

application and analgesic use and an orthopedics outpatient follow-up.

- Give the patient instructions about splint care, crutch use, range-of-motion exercises, weight-bearing status, warning signs for neurovascular impairment and compartment syndrome and follow-up.
- The patient can begin exercises when the pain subsides and can return to full activity when full pain-free motion and equal strength are attained in both limbs.

# Ankle

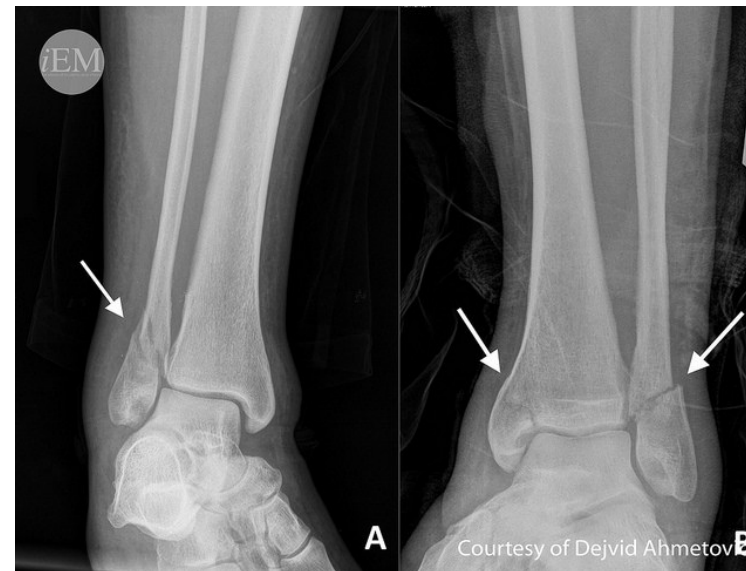
## Case Presentation

*A 25-year-old male presented to the emergency department with right ankle pain after a fall during a soccer game. His medial right ankle was swollen,*

•Soft tissue injury

and palpation revealed tenderness on the medial ligaments and the medial malleolus. He could not bear weight on his right foot. Anteroposterior and lateral ankle x-ray revealed a lateral and medial malleolar fracture (see Image 12.14) involving the joint. The patient was admitted to the orthopedics ward for elective surgical repair.

**Image 12.14** Fracture of fibula and fracture of medial malleolus



## Critical Bedside Actions and General Approach

These steps are the same as those mentioned above in the topic “hip.”

## Differential Diagnosis

The patient may have one or more of the following.

- Fracture (distal tibia, distal fibula, talus, calcaneus)
- Ankle dislocation
- Neurovascular injury

## History and Physical Examination Hints

- Note systemic illnesses, known metabolic disorders and medications, as mentioned above in the topic “hip.”
- Visual inspection and palpation: mentioned above in the topic “hip.”
- Check neurovascular status.
- Evaluate weight-bearing ability only if clinical suspicion of a fracture is low.
- The patient with a sprain may complain of hearing a “snap” or a “pop” at the moment of injury. Examine the joint for abnormal motion.
- Examine the proximal fibula in all medial ankle injuries. A medial ankle disruption (deltoid ligament tear or medial malleolar fracture) can cause complete tearing of the tibiofibular syndesmotic ligament and fracture of the proximal fibula (Maisonneuve fracture).



- See Images 12.15 and 12.16 for ankle open fracture plus dislocation.

**Image 12.15**



**Image 12.16**



- See [video](#) for ankle examination
- Achilles tendon may be ruptured in patients exhibiting posterior ankle pain after a shortfall or jump onto a slightly plantar-flexed foot. The Thompson test is used to assess the integrity of this tendon. (see [video](#))

## Emergency Diagnostic Tests and Interpretation

- The blunt ankle trauma evaluated within 48 hours of injury, the Ottawa Ankle Rules (OAR) can be used to determine necessity of x-rays. The OAR does not apply to the hindfoot or forefoot. Finally, the OAR is not applicable to intoxicated patients, patients with head injuries, multiple injuries, or diminished sensation related to neurologic deficits.
- Views of the ankle should include AP, lateral, and mortise views. The mortise view allows a reasonably good image of both the mortise and the talar dome.
- OAR [video](#)
- Plain radiography may miss subtle ankle fractures. If plain radiography is negative, but there is suspicion of a fracture, other imaging modalities or orthopedic consultation is advisable.

## Emergency Treatment Options

Ankle Dislocations: See video for reduction maneuvers. Reassessment of the neuro-vascular status, splint immobilization, ankle elevation, and post-reduction radiography should follow. Watch this [video](#).

Ankle fractures: Displaced intraarticular fractures require surgery.

Achilles Tendon Rupture: Splint the leg in plantar flexion; arrange orthopedic follow up as an outpatient.

Sprains: Application of ice, elevation, and analgesia are recommended. Nonsteroidal anti-inflammatory drugs (NSAIDs) are effective in many patients.<sup>3</sup> Immobilization of the limb for the first 48 to 72 hours provides protection and comfort. For complete or nearly complete ligamentous disruption, orthopedic consultation is mandatory.

A neurovascular injury requires urgent surgical evaluation.

## Special Populations

Treatment options are mostly the same for children, elderly and pregnant patients.

## Disposition Decisions

- Ankle dislocations and most ankle fractures should be admitted to the orthopedic ward. Consult an orthopedic surgeon.
- Soft tissue injuries can be discharged with the recommendation of ice application, elevation, immobilization, and analgesic use.

**References and Further Reading**, click [here](#)

# Pelvic Injuries

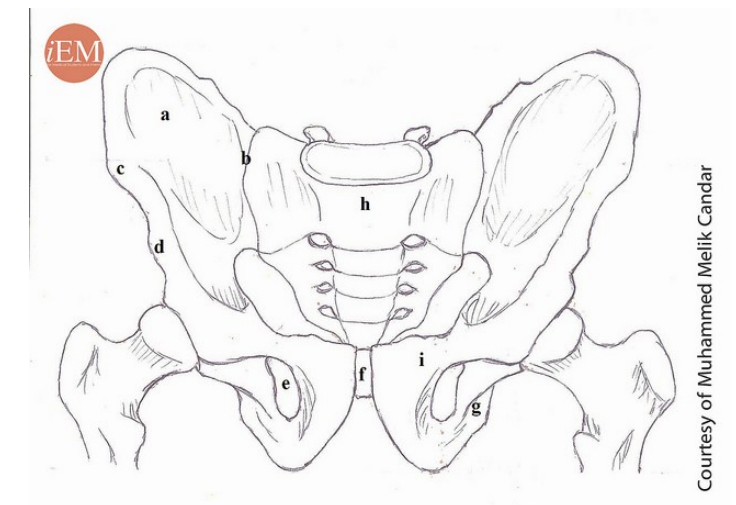
by Sercan Yalcinli

## Introduction

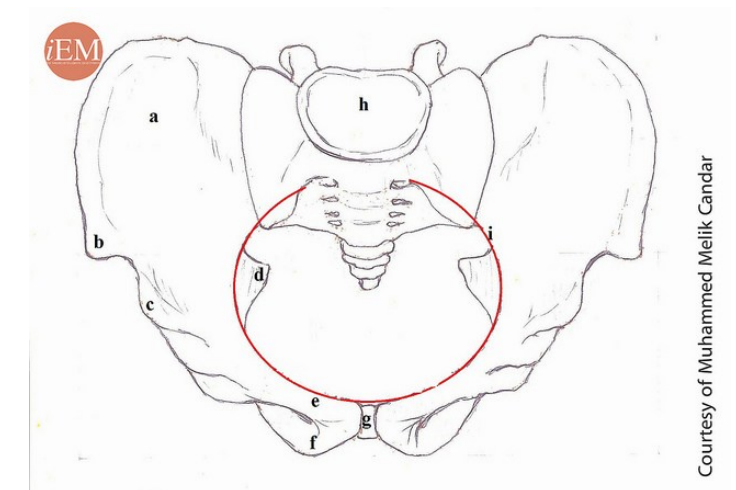
Pelvic fractures constitute 1-3% of pelvic skeletal fractures. In younger adults, a pelvic fracture is generally due to high energy pelvic injuries secondary to motor vehicle injuries, pedestrian injuries, falls from height, motorcycle accidents and crush injuries. In the elderly, it may occur following a fall from a sitting position. Pelvic trauma-related mortality is between 3-20%. It reaches 40-50% in hemodynamically unstable patients.

The pelvis is composed of three bones: Two innominate bones and sacrum. Ilium, ischium and pubis bones form the innominate. (See Illustration 12.3 , 12.4, 12.5)

**Illustration 12.3** Anterior view of pelvic bones



**Illustration 12.4** Superior view of pelvic bones





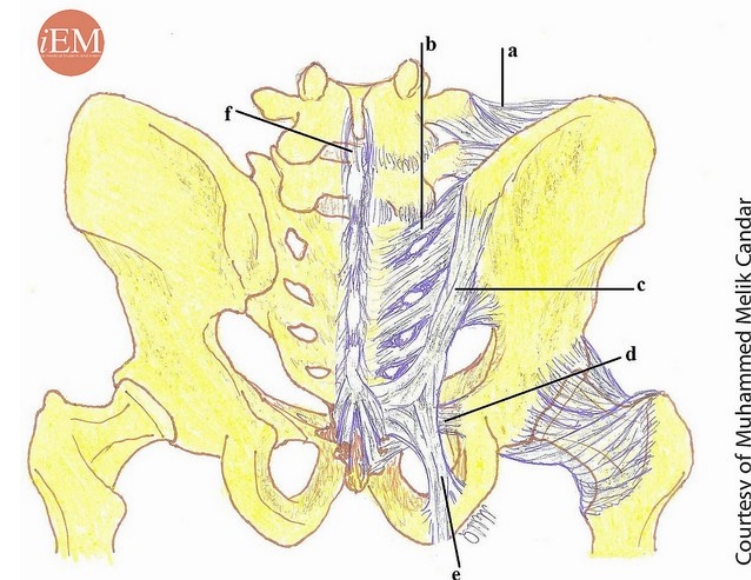
**Illustration 12.5** Posterior view of pelvic bones



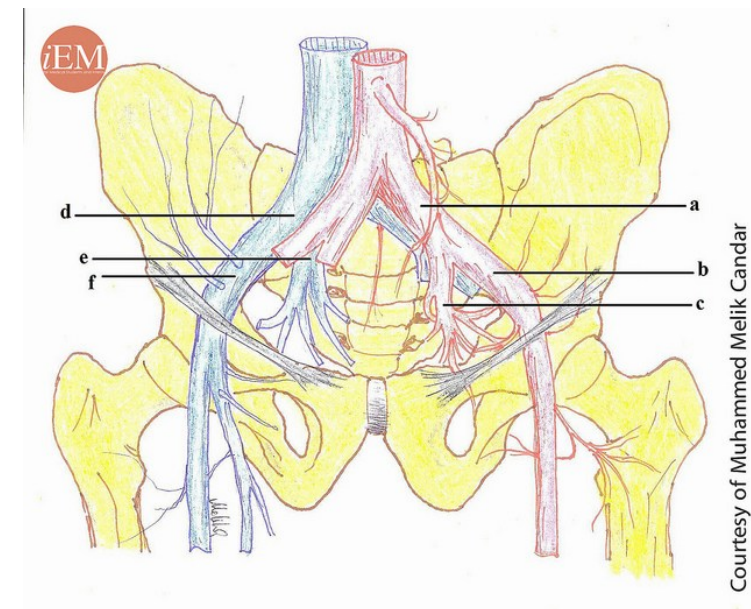
The posterior sacroiliac, sacrotuberous and sacrospinous ligaments located between the sacrum and two innominate bones provide the pelvic stability. Symphysis pubis supports pelvis frontally. (See Illustration 12.6)

The pelvis has a complex vasculature. Iliac arteries and main veins are close to both sides of the sacroiliac joints. (See Illustration 12.7) The thin-walled venous structures have limited contraction capabilities. Therefore, patients with pelvic fractures may have life-threatening bleedings.

**Illustration 12.6** Pelvic ligaments



**Illustration 12.7** Pelvic vessels



Three bones form the acetabulum. The ilium forms the upper boundary; ischium forms the posterior part and ilium and

**Illustration 12.8** Acetabulum anatomy



pubis form the anterior part of the acetabulum. (See Illustration 12.8)

Cauda equina courses through the sacral spinal cord and leaves at the sacral spinal foramina to form the lumbar and sacral plexus. Lumbosacral plexus is the thickest peripheral nerve of the body and

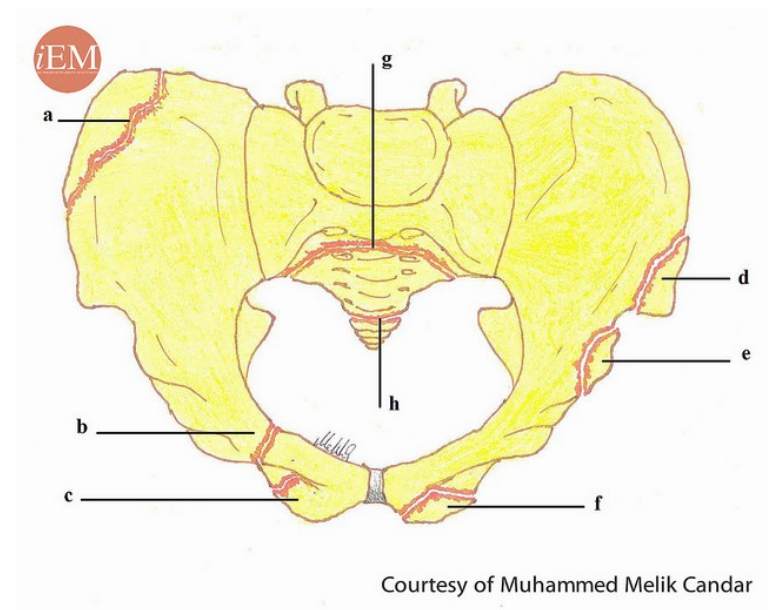


is frequently injured in posterior hip dislocation with acetabular fracture. Watch this [video](#) for detailed information.

Pelvic fractures are divided into three groups:

- a) pelvic fractures which cause separation of pelvic ring,
- b) no separation of pelvic ring with one bone fracture (see Illustration 12.9) and
- c) acetabular fractures.

**Illustration 12.9** Nondisplacement fractures of pelvis

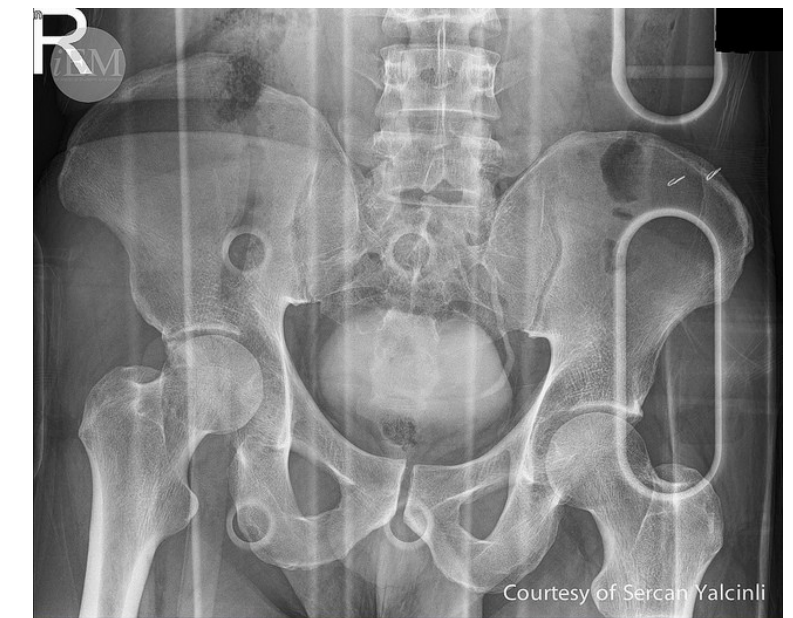


Acetabular fractures are often associated with the femur fracture, hip fracture and dislocations, and knee injuries. Posterior wall fracture is the most common acetabular injury and is usually associated with posterior dislocation of the hip. Posterior hip dislocation is generally associated with sciatic nerve injury. Watch this [video](#) for detailed information. (See image 1, 2, 3 )

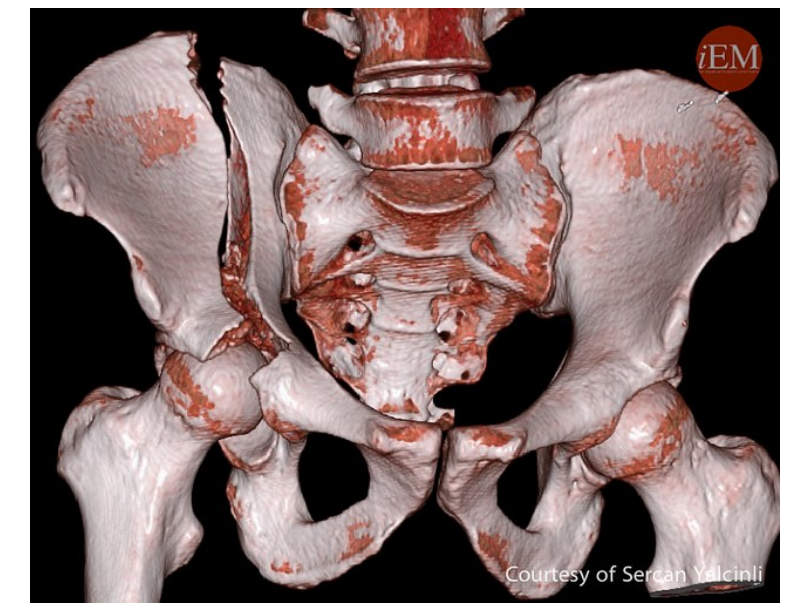
**Image 12.17** Posterior acetabular fracture associated with posterior dislocation of the right hip



**Image 12.19** Iliac and anterior column fracture of acetabulum



**Image 12.18** Iliac and anterior column fracture of acetabulum. 3D reconstruction in the CT of of the patient with image 12.19.





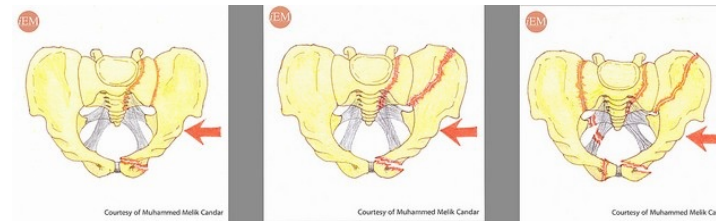
Several classifications are used to identify pelvic fractures (see [video](#))

Young-Burgess Classification classifies fractures according to the direction of the force that caused the injury and the mechanisms of injury. There are four different fracture models according to this classification system:

- lateral compression (LC),
- anteroposterior compression (AP),
- vertical scissor (VS) and
- combined mechanisms.

Lateral compression is the most common mechanism. It corresponds to 50% of the injuries. An example is a side impact of a motor vehicle to a pedestrian (see Illustration 12.10, image 12.20).

### Illustration 12.10 Lateral compression fractures of pelvis



Left to right. Type 1, Type 2, and Type 3. Type 1 includes sacral compression fracture on ipsilateral side. Type 2 is a sacral injury with disruption of posterior sacroiliac ligaments. Iliac wing fracture on impact side can be seen. Type 3 includes Type 1 and 2 injuries on impact side with open book fracture/injury on contralateral side.

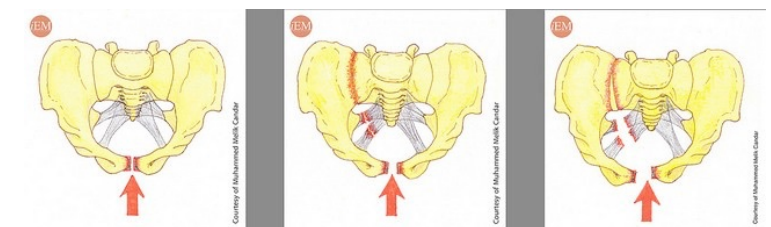
### Image 12.20



Lateral compression type 3 injury; inferior and superior pubic rami fractures on impact side and contralateral sacroiliac widening

A P compression or open book injury is the second most common mechanism and corresponds to 25% of injuries. An example is a frontal impact of the motor vehicle. (See Illustration 12.11) (See image 12.21 and 12.22)

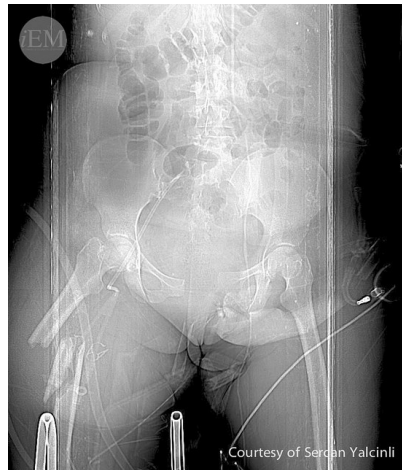
### Illustration 12.11 Anteroposterior compression fractures of pelvis



Anteroposterior compression fractures of pelvis. Left to right Type 1, 2, and 3. Type 1: symphysis pubis diastasis less than 2.5 cm, ligaments are stretched (anterior sacroiliac, sacra-tuberous, sacrospinous) but intact. Type 2: Symphysis separated more than 2.5 cm. ligaments (sacra-tuberous, sacrospinous) are disrupted. Sacroiliac ligaments are intact. Type 3: Symphysis separated more than 2.5 cm and all ligaments are disrupted.

### Image 12.21

Antero-posterior compression type 2 injury with right femur bone fracture



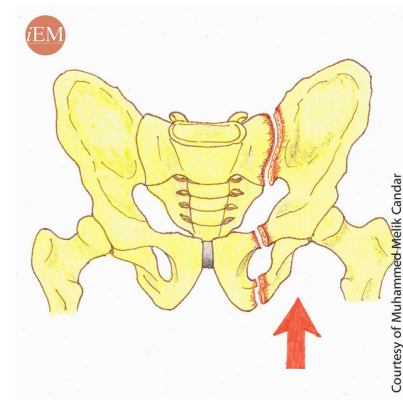
### Image 12.22 Type 3 injury



The least common is VS as it generates 5% of the injuries. Falls from heights are examples. (See Illustration 12.12, image 12.23)

### Illustration 12.12

Vertical compression injury. Pubis and sacroiliac joints are disrupted.



### Image 12.23 Left iliac wing, acetabulum and inferior pubis fracture of pelvis



Coexistence of other injuries constitutes 20-25% of injuries.

Tile classification is about the mechanical stability of the pelvis.

Type A – Stable pelvic ring injuries, posterior stability is intact: Avulsion fractures, isolated iliac wing fractures, isolated pubic rami fractures, transverse fractures of sacrum or coccyx.

Type B – Partially stable pelvic ring injury (incomplete disruption of the posterior pelvis) rotationally unstable, vertically stable: Open-book fractures, lateral compression fractures, double rami fractures and posterior injury

Type C – Unstable pelvic ring injury: Vertical shearing fractures, rotationally and vertically unstable.

Tile classification system predicts the need for surgical intervention. Young and Burgess determines the pattern of the fracture and predicts the chance of associated injuries and mortality risk.

## Case Presentation

*A 38-year-old male presents to the emergency department following a motor vehicle accident. The patient has left femoral and hip pain. His vitals are as follows: Blood pressure 100/60 mmHg, heart rate 108 beats per minute, pulse oxygen saturation at room air 99%. His physical examination reveals suprapubic tenderness, limitation of motion in the left hip joint, pelvic tenderness and hemorrhage at urethral meatus. Point-of-care ultrasonography shows no intraabdominal free fluid. Plain pelvic radiography and retrograde urethrography show superior pubic ramus fracture, sacral fracture, 3 cm displacement of the symphysis pubis, left femur bone fracture and urethral injury, respectively. Computerized tomography confirms retroperitoneal hemorrhage. The patient is brought to the operating room.*

## Critical Bedside      Actions and General Approach

In multi-trauma patient, start with general trauma care including ABC.

Mechanical stabilization and immobilization of the patient are important because they reduce the risk of bleeding and secondary organ injuries.

Consider other organ injuries, especially with unstable pelvic fractures (e.g., intraabdominal injuries, gastrointestinal tract injuries, genitourinary injuries, major vascular injuries, and neurological injuries)

- Check vital signs
- Physical examination
- Take medical history
- Learn mechanism of injury
- Ensure an iv line (except lower extremity)
- Type and crossmatch
- Order necessary imaging, labs, etc..
- Determine the need for operation and type of pelvic fracture, stable or unstable (mechanically and hemodynamically).



## Differential Diagnoses

- Abdominal pain in elderly
- Blunt abdominal trauma
- Hip dislocation
- Hip fracture

## History And Physical Examination Hints

The mechanism of injury plays a vital role in identifying pelvic fracture risk and determining the severity of the fracture. Low-energy injuries (such as falling on the ground) typically lead to a stable injury. High-energy injuries (such as motor vehicle accidents) increase the risk of unstable pelvic fractures and other organ injuries.

The direction of the force may give an idea about the type of injury. Antero-posterior forces may lead to open book injuries (such as motor vehicle accidents). The pelvic floor generally remains intact while lateral forces (side impacts) lead to injuries of the posterior ligaments. Vertical

forces (such as falls from heights) may lead to damage to the ligaments and pelvic floor that lead to significant instability in the posterior pelvis.

Patients should also be questioned for bladder tenderness, last urination and defecation time, last oral intake time, medical history, drugs and allergies, menstruation time and pregnancy status of females.

Rotation of the iliac wings indicates a severe pelvic fracture on inspection. Differences in limb length may be associated with hip injury or unstable or displaced hemipelvis. Careful inspection of the skin and skin folds is important for the detection of open fractures. Perineal ecchymosis or hematoma, Cullen's sign, and Grey Turner sign are late findings and may be visible due to retroperitoneal hemorrhage and intraabdominal hemorrhage on the periumbilical or flank section. (Image 12.24 and 12.25)

**Image 12.24** Cullen's Sign.



By Herbert L. Fred, MD and Hendrik A. van Dijk – <http://cnx.org/content/m14904/latest/>, CC BY 2.0, <https://commons.wikimedia.org/w/index.php?curid=5038484>

**Image 12.25** Gray Turner sign



By Herbert L. Fred, MD and Hendrik A. van Dijk – <http://cnx.org/content/m14942/latest/>, CC BY 2.0, <https://commons.wikimedia.org/w/index.php?curid=5038419>

In conscious patients without distracting injury, tenderness on palpation between the symphysis pubis, sacrum and the

sacroiliac joint may be a symptom of pelvic injury. Manipulation of the pelvis should be kept minimum because of the increased risk of hemorrhage due to the break down of clots that formed around the pelvic fracture. Hence, in an unstable pelvic fracture, the recurrent physical examination is contraindicated.

Presence of blood in the penile meatus should be noted. A digital rectal examination should be made to reveal the anal sphincter tone, prostate position and integrity, presence of mucosal disruption and bleeding caused by bone ends. Also, a digital vaginal examination should be done to detect open fractures in females.

Pulse and motor and sensory examination of the lower extremities should be evaluated.

## Emergency Diagnostic Tests and Interpretation

### Plain Radiography

ATLS guidelines recommend plain pelvic radiography for patients who have signs

of pelvic injury on physical examination, severe trauma mechanism, suppressed awareness or distracting injury.

The method for evaluation of plain pelvic radiography is described in [here](#).

Sacral fractures and sacroiliac joint injuries may not be seen on AP view. Inlet and outlet x-ray views increase sensitivity and specificity for the diagnosis of pelvic fractures in patients who have posterior pelvic tenderness but normal findings in anterior evaluation. (see image 12.26, 12.27, and 12.28)

**Image 12.27** Inlet view



**Image 12.26** Inlet view



**Image 12.28** Outlet view





**Image 12.29** Outlet view



Avulsion fractures of the L5 transverse process, avulsion fractures of adhesion places of sacrospinous and sacrotuberous ligaments, avulsion of lower lateral lip of the sacrum and vertical sacral fractures that extend to medial part of sacral foraminae may show an unstable pelvic fracture exclusively.

### Computed Tomography

- CT is the gold standard for evaluation of pelvic injuries.

- CT should be used when the clinical suspicion is high, but the plain pelvic radiograph is negative.
- CT identifies secondary injuries in patients with pelvic fractures on x-ray.
- It is preferred in suspected acetabular fractures.
- Contrast-enhanced CT gives useful data for evaluation of soft tissue injuries, vascular injuries, and pelvic hematoma.
- The presence of arterial bleeding is 80-90% recognizable with CT.

### Emergency Treatment Options

There is no standardized protocol for the treatment of pelvic injuries. Treatment options should be based on the hemodynamic status, the severity of trauma, the mechanism of injury, the type of fracture, and concomitant injuries.

### Resuscitation

- All critically ill patients should be given oxygen and intravenous fluids.
- Lower extremity veins should not be preferred as an intravenous line in patients with severe pelvic fracture because of the risk of leak into the retroperitoneal space.
- Opioids may be given for pain control.
- Antibiotics should be given for patients with bowel rupture, urogenital injury, and an open fracture.
- Tetanus prophylaxis is applied to appropriate patients.
- Crystalloid fluids and blood products may be required in patients with a pelvic injury.
- Open book injuries, fractures that cause separation of more than 0.5

cm in the pelvic ring, and fracture findings that include displacement at symphysis pubis or obturator ring may need the blood transfusion.

- Hemodynamically unstable patients, due to hemorrhagic shock caused by trauma, should be treated considering the ATLS guidelines.

## Control of Hemorrhage

Treatment choice should be selected according to the capacity of the health center with an emergency physician, an orthopedic surgeon and an interventional radiologist to take control of pelvic hemorrhage.

## Mechanical stabilization

- The bed linen is wrapped tightly around the pelvis as a simple non-invasive technique. Please see videos [1](#) and [2](#).
- Sheets must be wrapped to pass through the center of the trochanters instead of iliac crests.

- Open book injuries get the most benefit from bed linen wrapping method.
- This maneuver may aggravate the degree of the displacement in lateral compression injuries because of the internal rotational strain.
- External fixation and extraperitoneal packing may be preferred by the orthopedic surgeon as invasive treatment options.

## Angiographic Embolisation

Posterior pelvic ring injuries are associated with the most severe hemorrhages. The majority of pelvic bleeding has the venous origin. Arterial hemorrhages account for 10-15%.

Shock and death are associated more with arterial bleeding.

Angiography is indicated in patients with a major pelvic fracture who have resistant hypovolemia, although other resources for bleeding are under control .

Angiographic embolization is reported to be effective at controlling arterial bleeding, while external fixation is reported to be effective at controlling venous hemorrhage. However, it is difficult to determine the origin of the hemorrhage whether venous or arterial until angiography is applied.

## Complications

Timely intervention is crucial for prevention of complications.

Life-threatening hemorrhage, deformity, neurological and genitourinary injuries are complications that should be diagnosed and treated in pelvic traumas.

## Early Complications

- Hemorrhagic shock
- Urethral injury
- Bladder injury
- Vaginal laceration
- Rectal injury

- Perineal injury
- Limbo-Sacral nerve root injury

### **Late complications**

- Chronic pain
- Sexual dysfunction
- Shortening of extremity
- Malunion or nonunion

## **Pediatric, Geriatric, Pregnant Patients and Other Considerations**

The risk of hemorrhage is higher in pediatric patients. Child abuse should be considered. There is an increased risk of uterine rupture in pregnant patients. Consider deep vein thrombosis prophylaxis in non-ambulatory geriatric patients with stable pelvic fractures.

## **Disposition Decisions**

### **Admission criteria**

- Tile type B or C pelvic fractures
- Acetabular fractures
- Pelvic fractures with other system injuries

### **Discharge Criteria**

- Hemodynamically stable Type A pelvic fractures with no evidence of other system injuries.

**References and Further Reading**, click [here](#)

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by Ozge Can

## Cervical Spine Injuries

### Case Presentation

*A 19-year-old male presented to the emergency department with a fall from height. Emergency Medical Services Providers stabilized him on a backboard and cervical collar. On examination, his BP: 100/70 and HR: 60 GCS 13. There is swelling and ecchymosis right side of the neck but no vertebral step-off sign. His upper and lower extremities motor function was 2/5. The cranial and cervical computed tomography showed cervical dislocation and subarachnoid hemorrhage.*

### Critical Bedside Actions and General Approach

- Check vital signs
- Check immobilization



- Order monitorization and IV line
- Learn mechanism of injury
- Examine the vertebra
- Examine motor and sensory function
- Check exclusion rules
- Examine other injuries
- Order imaging and labs.

## Differential Diagnosis

- Spinal cord injuries
- Cervical spine injuries
- Cervical Ligamentous injuries
- Vertebral artery injuries
- Torticollis
- Cervical hematomas, masses

## History and Physical Examination Hints

- The examination must start with general trauma care. On physical examination, the emergency physician should

palpate the posterior structure of vertebrae behind the neck (Adam) after unfastening the patient's cervical collar and preventing the reflexive movement of the head with the other hand. Check the posterior neck for midline sensitivity, swelling, ecchymosis, step-off sign. Examine motor and sensory function.

- If the patient is fully conscious and has no posterior midline tenderness, the emergency physician may remove the cervical collar. Then rotate the head left and right, caudal and cephalad slowly and check if the patient is feeling any pain.
- Evaluate the exclusion criteria if the patient is conscious and has no posterior midline tenderness on examination. Nexus and Canadian C-spine rules are the main rule-out criteria of a cervical spine injury.

For nexus criteria, watch this [video](#).

For Canadian C-spine rules, watch this [video](#)

•Examine the motor and sensory function. Motor function is assessed from 0 to 5.

0 – is total paralyzed,

1 – is palpable contraction,

2 – motion with gravity,

3 – motion against gravity,

4 – motion is present but less power,

5 – normal power.

- To assess sensory function, examine deltoid muscle for C5, the thumb for C6, the middle finger for C7, and the little finger for C8.
- Check deep tendon reflexes (biceps, triceps).
- Spinal cord injuries may lead to neurogenic or spinal shock (See the shock and spinal cord injuries). Hypotension and bradycardia are the main symptoms of shock.

- Motorcycle accidents, falls from height and sports injuries are common causes of cervical spine injuries.
- Victims may be under effects from drug, alcohol, and unconsciousness associated with head trauma.
- Inspect for any other injuries such as maxillofacial and head injuries. Unconscious and vitally unstable patients with a head, abdominal and thoracic injuries should be considered to have a cervical injury.
- Learn the mechanism of injury. Cervical spine injuries are categorized according to mechanism into flexion, extension, and vertical compression.

### Flexion

- C1-2 atlantooccipital or atlantoaxial dislocation is caused by displacement of the head anteriorly and posteriorly. It is diagnosable by plain radiography. Atlantooccipital dislocation is a life-threatening injury and more frequent in children.

- Simple wedge fracture generally occurs anteriorly when longitudinal ligament pulls vertebrae body and ruptures due to flexion forces.
- Flexion teardrop is an unstable fracture caused by flexion forces. It is associated with severe ligamentous injury, anterior cervical cord syndrome and quadriplegia.
- Clay Shoveler's fracture is the stable fracture of the C7 spinous process.
- Spinal subluxation is characterized by a bone fracture with enlargement of interspinous and intervertebral space.
- Bilateral facet dislocation is associated with soft tissue, annulus fibrosis and anterior ligament injury.
- Simultaneous flexion and rotation forces may produce unilateral facet dislocation. C2 dens fracture.
- C2 dens fracture has three types:
  - Type 1: avulsion fracture

- Type 2: base of dens fracture
- Type 3: dens and vertebra fracture

### Extension

- C1 posterior arch fracture an unstable fracture of the atlas
- Hangman's fracture is the fracture of bilateral C2 pedicles

### Vertical compression fracture

- Jefferson Jefferson fracture is C1 burst fracture. It is characterized by widened predental space on open mouth odontoid X-ray.
- A burst fracture is mostly seen in lower vertebrae. Lateral views show the fracture best.

## Emergency Diagnostic Test and Interpretation

- Decide the need for imaging using exclusion criteria.

- Choose the best test for the patient according to your examination, findings and mechanism of injury.
- Imaging is indicated if pain and midline tenderness, neurologic deficit or intoxication/altered mental status is present.
- Computed Tomography (CT): Cervical CT is indicated if the patient is unconscious, the physical examination is unclear, the neurologic deficit is present or CT is planned for another injury (especially head maxillofacial trauma). However, current AJR guideline recommends CT scan in the presence of any violation of NEXUS or Canadian-C-spine rules.
- Because you may not have CT scan availability in some institutions, knowing how to interpret c-spine x-rays is important. Please see this [chapter](#). You will also see many c-spine injury samples in that chapter.

- Magnetic Resonance Imaging (MRI): Spinal Cord Injury without Radiographic Abnormality (SCIWORA) defines the presence of neurological deficit with no radiographic or computed tomographic features of spinal fracture or instability. Therefore, the presence of neurologic deficit necessitates MRI for the diagnosis of traumatic myelopathy.

## Emergency Management

- Trauma surveys should be applied any c-spine injury patients. See this [chapter](#). Following recommendations are specific to c-spine, not general trauma management.
- Immobilize the patient at the first contact. Watch this [video](#)
- Immobilization is the first step of management. If not done at the prehospital setting, immobilize the patient's neck, place the collar posteriorly with an assistant and fasten. Unfasten the collar as soon as possible to prevent complications after the spinal

injury is excluded by clinical or radiologic means.

- Consider full monitoring. Monitorize the patient for spinal and neurogenic shock or phrenic nerve paralysis.
- Apply sedation to prevent self-injury or other complications in agitated patients secondary to additional injuries or substance effects. (See [sedation chapter](#))
- Provide cervical immobilization with in-line stabilization during intubation. (See intubation indications in a trauma patient).
- In-line stabilization (video [1](#) and [2](#)): Have the assistant stand at the head of the patient and stabilize the patient's neck using both hands and prevent hyperextension.
- Intubation with video laryngoscope is recommended, if available.

- Continue immobilization until the imaging if the patient needs an emergent operation.

## Medications

Corticosteroid treatment for spinal cord injuries secondary to spine injuries has been shown that having many flaws, therefore is not recommended anymore.

Analgesics should be applied to awake patient.

## Pediatric, Geriatric, Pregnant Patients and Other Considerations

Some diseases may predispose a person to cervical injury. Rheumatoid arthritis may cause C2 transverse ligament rupture. Atlantooccipital dislocation is seen with Down syndrome

Patients with long-term corticosteroid use or osteoporosis are predisposed to fractures.

## Disposition Decision

### Admission

The patients with neurologic or spinal shock should be admitted to the ICU. The patients with unstable fractures should be admitted and/or operated immediately by neurosurgery.

## Discharge

Patients with stable fractures and no neurologic deficits may be discharged. Recommend Philadelphia or Miami collar to the patients with suspected ligament injury.

Refer the patients to neurosurgery clinic.

# Thoracic Spine Injuries

## Case Presentation

*A 40-year-old male presented to the emergency department after a motorcycle accident.*

*The patient reported chest pain. Blood Pressure: 130/80 mmHg. Heart Rate: 120 bpm. His Glasgow Coma Scale was 15. There was no midline tenderness on cervical examination. The respiratory sound was normal. His E-FAST examination showed pleural fluid. The chest x-ray revealed hemothorax. Thorax CT showed thoracic vertebrae spinous process fracture.*

## Critical Bedside Actions and General Approach

Please refer to cervical spinal injuries section.

## Differential Diagnosis

- Spinal cord injuries
- Thoracic Ligamentous injuries



- Vertebral artery injuries
- Rib fracture
- Pneumothorax
- Scapula fracture

## History and Physical Examination Hints

- Thoracic injuries mostly occur with high-energy mechanisms, namely, motorcycle accidents, fall from height and gunshot injuries.
- First, immobilize the patient if he is not on a backboard. A vacuum splint or scoop stretchers is useful. Logroll ([video](#)) the patient for examination. For logrolling, a leader and three assistants should be available.
- Examine the patient's vertebrae for midline sensitivity, swelling, ecchymosis, step-off sign. Additionally, check the motor and sensory function and deep tendon reflexes.

- A scapular injury is an indicator of high energy trauma. In case of scapular injury, consider a thoracic spine injury.
- Remember that spinal cord damage may lead to spinal and neurological shock.
- Learn the mechanism: Thoracic vertebra injuries are classified as flexion, extension, rotation, shear, distraction and axial compression injuries according to their mechanisms.
- Flexion injuries: occur with anterior compression. Instability is associated with the posterior ligament injury. Wedge fracture is an example. It is the most common fracture in the thoracic spine.
- Extension injuries: are anterior ligament, facet, laminar, spinous process injuries.
- Axial compression injuries: are burst fractures and occur with high-energy mechanisms.

• Flexion-rotation injuries: occur with a posterior ligament injury.

- Shear injuries: Posterior anterior, lateral listezis occur with ligament injury.

## Emergency Diagnostic Test and Interpretation

Patients who have vertebrae pain, midline sensitivity, bone deformity, neurologic deficit, more than 60 years old and high-energy mechanism requires imaging.

Anteroposterior (AP) and Lateral X-rays: AP images show lateral pedicles. Lateral images show subluxations, compression fractures (Image 1) and chance fractures.

Computed Tomography (CT): Patients with a neurologic deficit or altered mental status require CT.

Magnetic Resonance Imaging (MRI): Patients with the suspected ligament, disk or epidural space injuries require MRI.

## Emergency Management

- Check immobilization
- Remove backboard if there are no signs of injury. Prefer a sliding board instead of backboard when prolonged immobilization needed (i.e. the risk of injury continues).
- Provide full monitoring, especially in patients with spinal or neurogenic shock or phrenic nerve paralysis.
- Flexion restriction braces (Jewett or Knight-Taylor) is recommended if there is no stable angle fracture.

## Medications

Please refer to cervical spinal injuries section.

## Pediatric, Geriatric, Pregnant Patients and Other Considerations

Long-term corticosteroid use, malignancies or osteoporosis is predisposing factors for fractures.

## Disposition Decision

### Admission

Patients with spinal and neurogenic shock symptoms should be admitted to the intensive care unit.

### Discharge

Patients with stable fractures and no neurologic deficits may be discharged after the consultation with neurosurgery or orthopedic department. Referral to these clinics should also be planned.

# Lumbar Spinal Injuries

## Case Presentation

*A 70-year-old woman arrived in the emergency department after a fall from stairs. She had*

*a history of corticosteroid usage. Vital signs are normal. On her examination, she had pain on her back at the level of lumbar 2-3. The lateral X-ray showed an L2 compression fracture.*

## Critical Bedside Actions and General Approach

Please refer to spinal injuries section.

## Differential Diagnosis

- Spinal cord injuries
- Lumbar spine injuries
- Spinal epidural hematoma
- Paraspinal hematoma
- Retroperitoneal hematoma
- Renal injuries
- Soft tissue injuries
- Intraabdominal trauma

- Pelvic injuries

## History and Physical Examination Hints

Examination findings and history are similar to those of thoracic injuries.

Lumbar vertebrae fracture is the most frequent of all and may happen after relatively minor trauma.

Examine hip flexion, leg extension, ankle dorsiflexion, and toe extension to assess motor function.

Evaluate rectal tonus by a rectal exam in patients with suspected lumbar injuries.

Lumbar fracture types:

- Wedge fracture: Isolated anterior column fracture
- Burst fracture: Fracture of the anterior and middle column
- Flexion-distraction: injuries are most frequently seen in seat belt injuries. Fractures at T12-L1 junction is called chance fracture. Flexion-distraction

fractures may be associated with abdominal injuries.

- Transverse process fracture: is the most common lumbar spine fracture. An x-ray may miss most of the transverse process fractures. The abdominal or pelvic injury may accompany.

## Emergency Diagnostic Test and Interpretation

Please refer to thoracic spine injury section.

**Image 12.30** L1 burst fracture on lateral lumbar vertebrae view



**Image 12.31** Sagittal CT view of L2 compression fracture.



**Image 12.32** Sagittal MRI view of L2 compression fracture and extending to spinal canal.



Courtesy of Funda Karbek Akarca

## Emergency Management

Please refer to cervical injuries section.

## Medications

Please refer to the cervical thoracic injuries section.

## Pediatric, Geriatric, Pregnant Patients and Other Considerations

A lumbar fracture is rare in infants. Consider child abuse, especially in fractures with paralysis.

Consider epidural hematoma in geriatric patients on anticoagulation

## Disposition Decision

### Admission

Admit patients with shock and intraabdominal organ injuries to intensive care unit.

### Discharge

Patients with a simple transverse sacral fracture, isolated spinous fracture or isolated transverse process fracture may

be discharged. Patients with a simple wedge fracture and no neurologic deficit may be discharged with pain control. Discharge decisions should be taken with neurosurgery and/or orthopedic consultations. Referral to these clinics should also be planned.

**References and Further Reading**, click [here](#)



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by Meltem Songur Kodik

## Injuries of Shoulder

### Case Presentation

*A 50-year-old male was brought to the emergency department after he fell onto his outstretched arm while working in a construction pit. He complained of severe pain, instability and weakness on his right shoulder. His vital signs were normal. On the physical examination, the arm was in abduction and external rotation. Normal contour of the deltoid and acromion was lost. The patient was leaning away from the injured side and cannot adduct or internally rotate the shoulder even slightly without severe pain. His humeral head was palpable anteriorly, and all movements were limited and painful. Additionally, there was palpable fullness below the coracoid process, towards the axilla. The neurovascular examination was normal. Initial anteroposterior (AP) radiogram showed an*

*anterior glenohumeral dislocation. The dislocation was reduced in the emergency room.*

## Critical Bedside and General Approach

Preserving function, preventing infection and assuring perfusion of the limb should be the goals. Proper diagnosis and treatment are essential for establishing these goals.

- Check vital signs
- Learn mechanism of injury
- Take medical history
- Make an orderly and thorough examination
- Order necessary imaging and labs.
- Noncritical orthopedic injuries should be treated only after more threatening injuries have been addressed.

## Differential Diagnosis

Fractures: Proximal end of the humerus (the most frequently injured bone of the shoulder) [Image 12.33] , clavicle (80 % being middle-third fractures) [Image 12.34], scapula [Image 12.35].

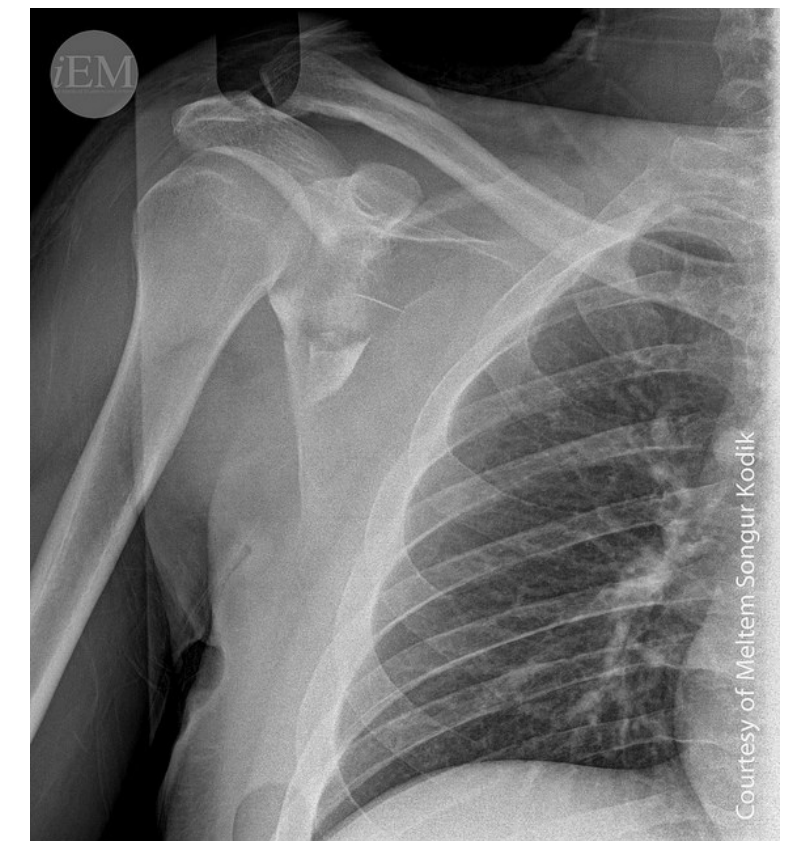
**Image 12.33** Proximal humerus fracture



**Image 12.34** Clavicle fracture



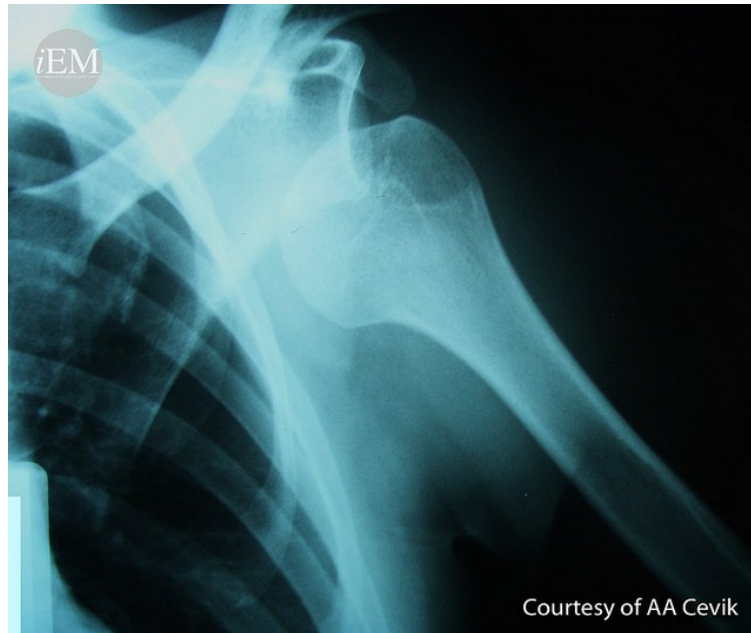
**Image 12.35** Scapular fracture



Dislocations: Glenohumeral (mostly anterior, rarely posterior, inferior and

superior) [Figure 4], followed by acromioclavicular, sternoclavicular.

**Image 12.36** Anterior humeral head dislocation



Soft tissue injuries: Impingement syndrome (Subacromial tendinitis and bursitis with rotator cuff tears, tendinitis of the long head of the biceps tendon)

## History and Physical Examination Hints

- Combination of pain, instability, stiffness and loss of power are the main complaints. The complaints usually start after trauma with acute pain.

Shoulder instability may be subtle or obvious subluxation or dislocation.

- The young athletes usually sustain anterior glenohumeral dislocation during athletic activities with rapid movements. The avulsion of the anteroinferior glenohumeral ligament is frequent. Characteristically, capsulolabral detachment occurs. (Bankart's lesion) In the elderly, a fall onto the outstretched arm is a more common mechanism of injury.
- With an anterior glenohumeral dislocation, the patients hold their arms in slight abduction with external rotation with the help of their opposite hand. The patients with an acute acromioclavicular sprain usually have a step-off at the joint and hold their arms to their side.
- Patients seeking medical care for their shoulder symptoms usually have a previous shoulder pathology. For patients with an obscure history of shoulder trauma, the patients' pain is

usually misreferred to as cardiac, biliary or abdominal pathology.

## Emergency Diagnostic Tests and Interpretation

For traumatic injuries, AP (45-degree lateral), transscapular lateral ("Y" view) [Image 12.37], and axillary lateral views are helpful. The true AP view provides a glenohumeral joint image without bony overshadow, so it is preferable over the standard AP view. The axillary lateral view is beneficial to evaluate the fractures of the glenoid fossa and the acromion or coracoid process.

The CT views are especially useful in determining scapular injuries. 3D reconstruction from chest CT provides requested soft tissue data details as well.

Bedside ultrasonography is particularly useful in clavicular fractures.



**Image 12.37** ransscapular lateral (“Y” view)



## Emergency Treatment Options

1. Initial Stabilization: Please refer to “Critical Bedside And General Approach” part. The incidence of neurovascular complications increases

with time. Therefore, the reduction of dislocation should be performed immediately. Before performing the reduction, the type of dislocation and any accompanying bone fractures should be shown radiographically.

2. Medications: For all reduction techniques, adequate sedation/analgesia or anesthesia is required. For many reductions, sedation with IV fentanyl (50 to 100 mcg) and IV midazolam (1 to 3 mg) is sufficient. Deep sedation with propofol or etomidate is required for some patients. Rarely, general anesthesia may be necessary. Injection of a local anesthetic intraarticularly is another good alternative and facilitates the reduction.

3. Analgesia:

- For children: Ibuprofen; Infants and Children <50 kg: Limited data available in infants <6 months: 4 to 10 mg/kg/dose every 6 to 8 hours; maximum single dose: 400 mg; maximum daily

dose: 40 mg/kg/day. Children ≥12 years: Refer to adult dosing.

- For pregnant patients: Paracetamol is still considered safe in pregnancy and should remain the first-line treatment for pain and fever. Dose: 325 to 650 mg every 4 to 6 hours or 1000 mg every 6 to 8 hours.
- For elderly: patients Acetaminophen 325 to 650 mg every 4 to 6 hours or 1000 mg every 6 to 8 hours.

4. Procedures:

### Shoulder Dislocations

Glenohumeral. Various techniques such as traction, leverage, or scapular manipulation can be used in combination for reduction. Some methods such as Hippocrates and Kocher maneuvers must not be used because of their high incidence of complications (axillary nerve injury, humeral shaft and neck fractures, and capsular damage). Watch [video](#) for one of the simple techniques.



One of the most frequently used adequate leverage technique is Leidelmeyer's external rotation method. While the patient is in supine position, the arm is gently adducted and the elbow is flexed to 90 degrees and gentle external rotation is applied until the reduction provided. The rate of success of this method is 80-90 %. There are other techniques such as Cunningham and scapular rotation techniques which requires almost no analgesia or sedation with high success rate.

## Fractures

- Clavicle: After closed reduction of the fracture, clavicular (figure-of-eight splint) splint should be applied.
- Scapula: To support the ipsilateral upper extremity, a sling immobilization is used.
- Proximal Humerus: A sling and swathe device is necessary for immobilization.

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

### Pediatric considerations

In the pediatric age group, closed reduction is similar to the adults. Under 10 years of age, shoulder dislocations are rare. Because of concomitant physeal (i.e., growth plate) fractures, an orthopedic consultation is recommended before reduction.

### Elderly considerations

In the elderly, anterior shoulder dislocation is more common; of those, 20% suffer redislocation and 60% have a cuff tear. To stabilize the shoulder the torn cuff should be repaired; patients with multiple redislocations usually require both procedures.

### Pregnant considerations

Procedural sedation in pregnant women provides relief from significant pain, distress or fear, and exposure to the medications used in procedural sedation is short and are relatively low. Such

features of this method provide no adverse pregnancy outcomes.

## Disposition Decisions

### Admission Criteria

If the closed reduction of shoulder dislocation fails, or when the patients have a neurovascular compromise, the patient may need admission for reduction under general anesthesia or open reduction.

### Discharge Criteria

After the reduction is accomplished, the shoulder is immobilized with velpeau bandage and the patient is discharged.

The patient is referred to an orthopedic surgeon within one week.

The patient should be informed if there is redislocation.

### Referral

Accompanying fractures of shoulder dislocations are Hill Sachs deformities (cortical depression in the humeral head created by the glenoid rim during

dislocation), Bankart lesions and fractures of greater tuberosity. In such circumstances, orthopedic referral is required.

# Injuries of Elbow

## Case Presentation

*An 8-year-old boy fell onto his left outstretched arm with a hyperextended elbow, while he was riding his bike to school. When he arrived at the ED, his left elbow was tender and swollen, and he was unable to move the forearm. His vital signs were normal. On physical examination, he was holding the upper extremity immobile in extension to the side. The*

*elbow appeared angulated and the upper extremity shortened. The neurovascular examination was normal. Radiogram, including anteroposterior (AP) and lateral films, revealed a supracondylar extension fracture. The prominence of the olecranon attached to the posteriorly displaced distal fragment is similar to that seen with posterior dislocation of the elbow. The fracture was stable after reduction and immobilized with a splint; elbow in a flexed position.*

## Critical Bedside and General Approach

These steps are the same as those mentioned above in the topic “shoulder.”

## Differential Diagnosis

### Fractures

#### Fractures of the distal humerus:

Supracondylar fractures [Image 12.38]; Extension type (mostly) and Flexion type

- Type 1: Minimal or no displacement
- Type 2: Slightly displaced
- Type 3: Totally displaced

Transcondylar, intercondylar, lateral condyle, and medial condyle fractures

Articular surface fractures: Capitellum, Trochlea, Epicondyle, Little Leaguer’s Elbow, Olecranon

Radial head and neck fractures

#### Dislocation and Subluxation:

Elbow: Posterior (Mostly) [Image 12.39],  
medial, lateral, anterior (Rarely)

Radial head (nursemaid' s elbow)

**Soft tissue disorders:** Epicondylitis  
(Tennis Elbow), Olecranon bursitis, biceps  
tendon rupture.

**Image 12.38** Supracondylar fracture.  
Type 2



© Courtesy of iEM for Medical Students and Interns (AAC)

**Image 12.39** Elbow dislocation



## History and Physical Examination Hints

Indirect trauma transmitted through the  
bones of forearm causes many of the  
elbow injuries. (e.g., FOOSH= Fall on  
outstretched hand )

Very few fractures or dislocations  
occur by direct blows.

Numbness or weakness distal to the  
injury should indicate the possibility of  
neurovascular injury.

Extension type supracondylar fracture  
has a characteristic S-shaped  
configuration of the arm held at the side.  
On the other side, flexion type  
supracondylar fractures leads the  
supporting of the forearm with the  
opposite hand where the elbow is flexed  
at 90°. When the prominence of  
olecranon is increased, it suggests  
posterior dislocation of the elbow or  
extension supracondylar fracture.  
Anterior dislocation or flexion  
supracondylar fracture leads to the loss  
of normal olecranon prominence.

When the prominence of olecranon is  
increased, it suggests posterior  
dislocation of the elbow or extension  
supracondylar fracture. Anterior  
dislocation or flexion supracondylar

fracture leads to the loss of regular olecranon prominence.

Gradual onset of dull ache at the elbow happens with epicondylitis. This pain increases with grasping and twisting of the elbow.

With the elbow in a 90° flexion, the radial head, tip of the olecranon and the lateral epicondyle normally form an equilateral triangle. Fracture of the radial head, olecranon or the lateral epicondyle alters this relationship.

A complete neurovascular examination should be made for the elbow and distal extremity. This examination should include: sensation and strength tests of the median, radial, ulnar, and musculocutaneous nerves, deep tendon reflexes of the biceps (C5), brachioradialis (C6), and triceps (C7), palpation of the brachial, radial, and ulnar pulses, the range of motion and strength of the elbow should be examined.

## Emergency Diagnostic Tests and Interpretation

Plain radiographs should be obtained in three views of plain x-rays – AP, lateral, and lateral oblique are necessary. AP view shows the epicondyles (medial and lateral) and the articular surfaces. (radiocapitellar and ulnotrochlear) The lateral view provides the relation of the bones of the distal humerus and proximal forearm. The radiocapitellar joint, medial epicondyle, radioulnar joint, and coronoid process view with the lateral oblique.

Fat pad sign: With a history of known or suspected trauma of the elbow, if there is an abnormal fat pad sign this should be considered as an indication of an occult fracture. Wide anterior fat pad, also, known as “sail sign” indicates an occult fracture. Posterior fat pad sign in an adult indicates radial head fracture; in children indicates supracondylar fracture. Watch this [video](#).

X-rays of the uninjured elbow help distinguish fractures from the normal

epiphyses and ossification centers. To identify fractures, physeal injuries and dislocations ultrasound is a useful tool. Children with lateral condyle fractures may benefit from an MRI.

## Emergency Treatment Options

### Initial Stabilization

Before taking radiographs, to prevent further injury, immobilization should be performed. Please refer to “Critical Bedside And General Approach” part in the topic “shoulder.”

### Medications

Please refer to the topic “shoulder.”

### Procedures

Supracondylar fractures:

- Nondisplaced fractures of children do not require immediate orthopedic evaluation. These patients may be referred for follow up within a week after splinting. The family should be informed to return if an unmanageable



pain or compartment syndrome happens.

- Displaced supracondylar fractures generally require open or closed reduction and percutaneous pinning for monitoring of pulses, nerve function and forearm compartments; the patient should be hospitalized. The patient is discharged with a splint or cast with percutaneous pins.

Transcondylar, intercondylar, condylar, epicondylar fractures, articular surface fractures: orthopedic consultation is necessary for all these.

Elbow dislocations: to avoid vascular damage, reduce immediately, flex to 90° and place a posterior splint.

Radial head fracture: for all fractures for 24 to 48 hours a sling or posterior splint and cold application, analgesic etc. applied, and the patient should be referred to an orthopedic surgeon.

Radial head subluxation: for reduction of RHS, there are two techniques –

supination/flexion and hyperpronation. The latter is more successful for the reduction and may be less painful.

Epicondylitis: Once a clinical diagnosis of epicondylitis is made, the initial treatment consists of activity modification, counter force bracing, nonsteroidal anti-inflammatory drugs (NSAIDs) (if not contraindicated) and physical therapy. Severe cases can be splinted.

## **Pediatric, Geriatric, Pregnant Patient, and Other Considerations**

### **Pediatric**

In children younger than ten years of age, posterior elbow dislocation is the most common. Due to the different stages of ossification and predominance of cartilage, there is difficulty in interpretation of pediatric elbow X-rays. In the pediatric elbow, the six ossification centers are shown numbered in the order of appearance: Capitellum, radial head, internal (medial) epicondyle, trochlea,

olecranon, external (lateral) epicondyle (mnemonic: CRITOE).

## **Disposition Decisions**

### **Admission Criteria**

- Open fractures accompanying vascular injuries
- When an operative reduction or internal fixation is indicated
- All the patients with gross swelling and ecchymosis should be hospitalized for observation to monitor and decrease the risk for compartment syndrome.

### **Discharge Criteria**

- Reduced dislocations and stable fractures with none of the above features
- Splinted patients with an arrangement of orthopedic follow up in 24-48 hour
- Simple soft tissue injuries

### **Referral**

For a close follow-up, all fractures discharged from ED should be referred to an orthopedic surgeon.

# Injuries of Wrist

## Case Presentation

A 63-year-old woman fell on her outstretched hand while she was replacing a bulb at home. She presented to the ED with tenderness, severe pain, swelling, numbness, and coldness in the region of the wrist. On physical examination, she had swelling and “dinner-fork” deformity. The neurovascular examination was normal. On the PA and lateral views of the wrist, a distal

*radius fracture, called Colles’ fracture is evident. The emergency physician performed an early anatomic reduction with the restoration of radial length and correction of the dorsal angulation to a neutral position.*

## Differential Diagnosis

### Distal radius and ulna injuries:

- Colles’ Fracture (the most frequent): Transverse fracture of the distal radial metaphysis, which is dorsally displaced and angulated. [Image 12.40]
- Smith’s Fracture (Reverse Colles’ Fracture): Transverse fracture of the distal radial metaphysis with associated volar displacement and angulation.
- Barton’s Fracture: Oblique intra-articular fracture of the rim of the distal radius with associated displacement of the distal radial fragment.

**Image 12.40** Colles’ Fracture



- Hutchinson’s Fracture (Chauffeur’s fracture): Intra-articular fracture of the radial styloid.
- Distal radioulnar joint disruption
- Pediatric fractures of the distal radius: Torus [Image 12.41], Greenstick, complete fractures [Image 12.42]

**Image 12.41** Torus fracture



**Image 12.42** Complete shaft fracture of radius and ulna



### Carpal injuries:

Fractures: Scaphoid [Image 12.43], lunate, triquetral, pisiform, hamate, trapezium, capitate, trapezoid

Clinical features: Carpal instability, midcarpal and intercalated segment instability, radiocarpal instability

Soft tissue injuries: Carpal tunnel syndrome (due to distal radius fractures and repetitive strain), de Quervain's Disease and intersection syndrome

**Image 12.43** Scaphoid fracture





## History and Physical Examination Hints

Falling on an Out-Stretched Hand (FOOSH) is the most common mechanism of wrist injuries, with the wrist in extension.

Immature, weaker epiphyseal plate or metaphysis of the radius in children are more likely to sustain injuries, sparing the still-cartilaginous carpal bones.

Young adults with active lifestyles are more likely to be injured with greater forces.

In the elderly, especially in women with some degree of osteoporosis, distal radial metaphysis is more fragile resulting in Colles fracture.

“Anatomic snuffbox” on the dorsum of the wrist is an important landmark. Because the scaphoid is palpable with its triangle by styloid, extensor pollicis brevis tendon and the extensor pollicis longus tendon. Tenderness in this area may indicate a scaphoid fracture.

The examination should include assessment of neurovascular status motor and sensory function of the median, radial and ulnar nerves. Because acute median nerve compression is a common occurrence, the sensation of thumb and index fingers is important, especially with severely displaced fractures. In all injuries to the wrist, radial and ulnar pulses should be evaluated.

## Emergency Diagnostic Tests and Interpretation

Standard views of the wrist include posteroanterior (PA), lateral, and oblique views. MRI and CT provide the diagnosis of the radiographically occult fractures.

## Emergency Treatment Options

Initial Stabilization: Please refer to “Critical Bedside And General Approach” part in the topic “shoulder.”

Medications: Please refer to the topic “shoulder.”

Procedures:

Distal radius and ulna fractures:

- Colles’ and Smith’s fractures: Closed reduction and immobilization in a sugar-tong splint. Because the Smith’s fracture, unlike Colles’ fracture, is more unstable, it requires operative intervention and splint reduction.
- Barton’s Fracture: Since those fractures do not benefit close reduction, they should be referred to an orthopedic surgeon immediately.
- Hutchinson’s Fracture: If the fracture is non-displaced, a sugar-tong splint immobilization is sufficient. In such cases, a short arm cast is a definitive treatment. On the other hand, displaced fractures accompanied with a scapholunate ligament injury, open or closed reduction and fixation is necessary. Since many ligaments of the wrist are attached to the styloid process, it should be reduced accurately for wrist function.



Distal radioulnar joint disruption: A long arm cast is applied after closed reduction

Pediatric fractures of the distal radius: A short arm splint is required for immobilization.

Carpal injuries: A short arm splint with a thumb spica is required for immobilization.

Carpal tunnel syndrome: The wrist is splinted in a neutral position and cortisone injections are given additionally into the carpal tunnel.

De Quervain's Disease: for mild and moderate forms conservative measurements such as rest of the arm in elevated position, splinting, NSAIDs and corticosteroid injections into dorsal extensor compartment of the wrist may be sufficient.

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

### Pediatric

The most common fractures in children and adolescents are the distal radial fractures (44). Commonly growth plate injuries occur with the distal radius fractures leading to physeal injuries (45). The most important aspect of these physeal fractures is premature closure and growth arrest of the injury site.

### Geriatric

Distal radius fractures in older patients should be screened for osteoporosis. Also, distal radius fractures in an senior man is an early and sensitive marker of skeletal fragility (47). Subsequent fractures are prevalent and treatment of underlying osteoporosis in this population group is required.

## Disposition Decisions

### Admission Criteria

- Open fractures

• Fractures with compartment syndrome or neurovascular compromise

- Fractures needing immediate operative management or general anesthesia for reduction
- Fractures associated with soft tissue complications
- Fractures associated with circulatory deterioration in the hand

### Discharge Criteria

- Appropriate reduction and immobilization
- Orthopedic follow-up should be arranged
- Pain control measures should be taken adequately
- Cast or splint care instructions should be given at discharge and should be assured the patient understands them.

- After ED treatment, the documentation of intact neurovascular function is performed.

## Referral

For a close follow-up, all fractures discharged from ED should be referred to an orthopedic surgeon.

**References and Further Reading**, click [here](#)

## Chapter 13

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# Selected Infectious Problems



# Epiglottitis

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by Kuan Win Sen

## Case Presentation

*A 62-year-old man presents to the ambulatory area of the emergency department complaining of sore throat, fever, and chills. He has history of type 2 diabetes mellitus, hypertension, and obesity. He was seen by the general practitioner (GP) 2 days prior and was prescribed thymol gargle and paracetamol. Further history reveals progressive difficulty and pain in swallowing, decrease in appetite, and worsening sore throat since the GP visit two days ago. On physical examination, he is alert, has a temperature of 39.1C, heart rate of 112 per minute, blood pressure of 136/74mmHg, respiratory rate of 18 per minute and oxygen saturation of 98% on room air. He speaks with a muffled voice and has drooling of saliva. There are no obvious findings in the oropharynx and no cervical lymphadenopathy. The rest of the physical examination is unremarkable.*



## Critical Bedside Actions and General Approach

Every patient who presents to the ED will receive initial bedside evaluation (ABC...), and vital signs check. Depending on the history and general look of the patient, our first priority is to evaluate some symptoms and signs of impending airway obstruction. This evaluation can be done in the triage or inside the ED. If there are findings, the patient should be triaged to high acuity area. Airway equipment and monitoring devices should be placed to the patient. Depending on the patient's initial evaluation, any abnormality found in ABC evaluation should be treated immediately. However, suspicion of epiglottitis may change some management strategies such as involving airway specialists earlier.

## Differential Diagnoses

- **Peritonsillar abscess**
- **Diphtheria**
- Submandibular space infection (**Ludwig's angina**)

- Laryngotracheitis (**croup**)

## History and Physical Examination Hints

Have a high index of suspicion for epiglottitis in patients reattending ED for worsening sore throat. Severe sore throat with odynophagia, high fever, muffled voice ("hot potato voice") and drooling are common clinical features of epiglottitis. Patients with impending airway compromise would refuse to lie down for examination. Suspect epiglottitis if the severity of sore throat is out of proportion to findings in the oropharyngeal examination.

- Assess the soft palate for swelling and/or deviation of the uvula to exclude peritonsillar abscess (quinsy)
- Assess the sublingual and submandibular regions for swelling to exclude Ludwig's angina
- Assess the tonsils for grey pseudomembranes that are classically seen in diphtheria

Patients with croup have a "barking" cough and are generally comfortable in the supine position (watch the **video**).

## Emergency Diagnostic Tests and Interpretation

Obtain a lateral neck X-ray ("thumb" sign)

**Image 13.1**



*Typical findings of epiglottitis with enlarged epiglottis and aryepiglottic folds.*

*Case courtesy of Dr Maxime St-Amant, Radiopaedia.org. From the case rID: 26840*

Prepare for laryngoscopic visualization using a flexible nasolaryngoscopy (gold standard). Watch the [video](#).

Diagnostic laboratory investigations (complete blood count and blood culture) should not be performed in patients where suspicion of epiglottitis is high until the airway is secured.

## Emergency Treatment Options

Airway maintenance is paramount

- Provide humidified supplemental oxygen
- Consider prophylactic intubation to secure the airway in progressive disease
- Prepare the surgical airway set for the possibility of encountering a difficult airway
- Avoid supraglottic devices as they may compress the swollen epiglottis

Administer antibiotics (intravenous ceftriaxone 2g and intravenous clindamycin 600mg)

Other options such as Racemic epinephrine, eta-agonists, and corticosteroids have not been shown to be beneficial in epiglottitis treatment.

## Pediatric Considerations

Epiglottitis used to be more common in children with incomplete or lack of Hib immunization. However, the adult to child ratio has increased since the turn of the century.

Typical findings in children:

- Look toxic
- Reluctant to lie down
- Classical drooling, dysphagia, distress (3D's)
- Tripod position
- Sniffing position

Individualize examination according to the severity of illness

- those with classic symptoms of epiglottitis or stridor should have involvement of pediatric airway experts before attempts at visualization as there have been reports of cardiorespiratory arrest in children during these attempts

## Disposition Decisions

Intubated patients or those who require very close monitoring should be admitted to the surgical intensive care unit or high dependency unit.

**References and Further Reading**, click [here](#)

# Meningitis

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by Alja Parežnik

## Case Presentation

*A 55-year-old previously healthy woman presented with fever, headache, vomiting, and photophobia for three days. One week earlier, she started to complain about a sore throat and pain in the right ear. Neurological examination revealed diminished consciousness and neck rigidity. Lumbar puncture was performed and in CSF found  $>10.000$  leukocytes/mm<sup>3</sup>. Direct examination of CSF showed Gram-positive cocci in chains and culture yielded *S. pyogenes*. The patient had treated with Ceftriaxone (4 gr/day).*

## Introduction

Meningitis is an inflammation of the membranes of the brain and spinal cord. It can be related to infectious and noninfectious causes. The infection agent is usually bacteria or virus, and occasionally fungus. Additionally; physical injury, autoimmune disorders, cancer or certain drugs can cause meningitis .

## Pathogenesis

Bacteria can breach the blood-brain barrier (BBB) to infect the meninges by direct spread, or contiguous infection (from a source such as the sinuses or middle ear), trauma, neurosurgery, or indwelling medical devices. Nasopharyngeal colonization from infected droplets of respiratory secretions or distant localized infection (lungs, urine) with subsequent bloodstream invasion, are other sources of infection.

Pathogens causing meningitis can be spread in different ways:

- during birth from mother to her baby,

- through stool (enteroviruses),
- through coughing and sneezing,
- through kissing, sexual contact or contact with infected blood,
- from eating a specific food (*Listeria monocytogenes*),
- from rodents and insects (leptospirosis by mice, hamsters, rats and West Nile virus through mosquito bites).

## Etiology

The severity of illness and the treatment differ depending on the cause.

**Bacterial meningitis** is a life-threatening neurological and infectious emergency. It can lead to death within hours. Bacterial meningitis can lead to long-term problems, like hearing loss, vision loss, problems with memory and concentration, epilepsy, coordination, movement and balance problems, learning difficulties and behavioral problems. In community-acquired meningitis, *S. pneumoniae* is the most

common pathogen since routine immunization of infants with *H. influenzae* type B began in 1992. Table 13.1 presents the most common bacteria and their specification. *M. tuberculosis*, *S. aureus*, *Borrelia burgdorferi* and gram negative bacilli are among the rare causes.

**Viral meningitis** is much more common than bacterial. It tends to be less severe and usually recovers completely without specific therapy. Most common viral pathogens causing meningitis are;

- Enteroviruses (Coxsackie, echoviruses)
- Arboviruses (KME, West Nile),
- Herpes viruses (HSV-1,2, VZV, EBV, CMV)
- Others (mumps, HIV, parvovirus, rotavirus, etc)

**Fungal meningitis** is rare form and generally occurs only in immunocompromised people.



**Table 13.1** Main common bacterial pathogens in meningitis and their specifications

	NEISSERIA MENINGITIDIS	STREPTOCOCCUS PNEUMONIAE	HAEMOPHILUS INFLUENZE	LISTERIA MONOCYTOGENES
Age	Children, adults (living in crowded spaces)	Children, adults	Adults, not vaccinated children	older, newborns, pregnant women, immunocompromised
Vaccine	yes	yes	yes	
Associated diseases	Sore throat	Ear infection, sinusitis, pneumonia	Sinusitis	
Characteristics	Petechial rash, muscle pain and weakness	Rash, Neurological changes (seizures, focal)	Rash	

Original by author

### Critical Bedside Actions and General Approach

Stabilization of an unstable patient is the priority. Check the airway and breathing (respiratory rate, saturation) and give oxygen if needed. Check circulation (pulse, capillary refill time, urine output, blood pressure) and give fluids or medications if needed. Then, check disability (Glasgow coma scale or AVPU (alert, voice, pain, unresponsive), focal neurological signs, seizures, papilledema, glucose).

### Differential Diagnoses

- Encephalitis,
- meningitis,
- septicemia,
- brain abscess,
- subdural empyema,
- subarachnoid bleeding,
- tetanus,

• malaria,

- cancer of meninges,
- vasculitis of CNS.

### History and Physical Examination Hints

The classic triad with fever, neck stiffness and altered mental status is present in only 44% of cases. However, the absence of all of the triad almost eliminates the possibility of meningitis. More than 95% of patients have two out of four criteria: a headache, fever, neck stiffness and altered mental status. Some patients may show dislike of bright lights, rash, sleepy or difficulty to wake, or seizure. Babies may present with refusing to feed, irritable, high pitched or moaning cry, stiff body with jerking movements.

Some people are at the highest risk for developing meningitis. Risky age groups are children under age 5, teenagers from 16-25 years and adults over the age of 55. Certain medical conditions, such as

damaged or absent spleen, chronic disease or immune system disorders increase the risk. Traveling to areas where meningitis is common is another risk factor.

Search for meningismus sign using one or more of the following tests. In jolt accentuation test, the patient rotates his head horizontally at a frequency of two to three rotations per second. The test is positive if there is the exacerbation of an existing headache. The absence of jolt accentuation has a specificity of 97% for ruling out meningitis. Kernig's sign is the inability to straighten the leg when the hip is flexed to 90 degrees. Brudzinski's sign is positive when forced flexion of the neck elicits a reflex flexion of the hips. Both Kernig and Brudzinski have reported low sensitivity (5%) but high specificity (95%). Most common meningeal symptoms and their sensitivity are presented below.

- Fever >38 °C: 75-85%
- Stiff neck: 70-83%

- Altered mental status: 69%
- Headache: 87%
- Vomiting 35%
- Focal neurological exam: 23-33%
- Seizures 15-30%
- Kernig's sign 9%
- Brudzinski' sign 1%
- Jolt accentuation of headache 100%

Abnormal neurologic exam, photophobia, and lethargy are among the other related signs. Petechial rash, muscle aches, and weakness are characteristic for meningococcal meningitis.

Tests for confirming physical meningeal signs:

A) **Kernig's sign** is an failure to straighten the leg when the hip is flexed to 90 degrees because of the stiffness of the hamstrings. Watch the [video](#).

B ) **Brudzinski's sign** is neck stiffness causes a patient's hips and knees to flex when the neck is flexed. Watch the [video](#).

In sum, ask about typical clinical signs: fever, headache, photophobia, stiff neck, vomiting, and neurological changes. Previous diseases, associated diseases, family history, vaccination, allergy, social status, medications and traveling to other countries are also important. Pay attention to age, immunocompromised state, living in dorms, traveling to states with common meningitis infection, contacts with infected people.

In the physical exam, we look for the meningeal signs with different tests (jolt accentuation of headache, Kernig's sign, Brudzinski's sign) and neurological changes (AVPU, GCS).

## Emergency Diagnostic Tests and Interpretation

Routine blood work is often obtained but frequently unrevealing. Blood cultures should be drawn in all patients.

The gold standard of confirming meningitis is an analysis of **cerebrospinal fluid** (CSF) obtained by **lumbar puncture (LP)**. It should be obtained in all patients with suspected meningitis unless contraindicated. The first results are gram stain and latex agglutination tests. The culture of CSF later confirms the diagnosis. The combination of all three mentioned tests is proved to be more productive than any of the single test alone. Specific findings (white and red blood cell counts, glucose and protein levels) in CSF helps us to differentiate between the types of meningitis.

It is essential to perform LP as soon as possible. In some cases, LP is delayed due to imaging, limited resources, signs of severe sepsis or rapidly evolving rash, severe respiratory or cardiac compromise and significant bleeding risk. It is prudent to give empiric antibiotic therapy first. In pneumococcal meningitis, an approximate window to perform an LP after antibiotic administration is 4-10

hours. In meningococcal infections, it is only 1 hour.

**Computer tomography (CT)** has to be performed before lumbar puncture (LP) in order to exclude increased intracranial pressure (ICP) or alternate mass lesion when the patient has any of these criteria: immunocompromised state, history of CNS disease, new-onset seizure, papilledema, abnormal level of consciousness (GCS<12) or focal

neurologic deficit. **CSF analysis** is important, table 13.2 below shows the main characteristics of CSF in different types of meningitis.

**Table 13.2** Common CSF findings in different types of meningitis

INDEX	NORMAL	BACTERIAL	VIRAL	FUNGAL
WBC (white blood cells)	<5	>1000	<1000	<1000
Differential (neutrophils)	<15%	>80%	<15%	<15%
CSF glucose (mg/dL)	45-65	reduced	normal	reduces
CSF protein (mg/dL)	20-45	>250	50-250	>250
Opening pressure (cmH <sub>2</sub> O)	<20	Normal to high, typically 15-30	Normal to high	Normal to high
Gram stain		+		
PCR			+	

Quattromani EN, Aldeen AZ. Focus on: emergent evaluation and management of bacterial meningitis. American college of emergency physicians news. 2008 May[updated 2014]. <https://www.acep.org/Clinical---Practice-Management/Focus-On--Emergent-Evaluation-and-Management-of-Bacterial-Meningitis/>. Accessed April 18, 2016.

## Emergency Treatment Options

### Initial stabilization

Obtain venous access, give oxygen and fluids and resuscitate if necessary.

### Medications

Antibiotics are the cornerstone of treatment of bacterial meningitis. Administer empiric antibiotic therapy as soon as possible, before LP.

### Empirical antibiotic therapy options;

- Ceftriaxon/Ceftazidime 4g IV daily immediately (child 100mg/kg up to 4g)
- + Vancomycin 12,5mg/kg up to 500mg IV 6 hours if S.pneumonie or S.aureus is suspected, recent travel, risk of resistance
- + Ampicillin 2,4g IV 4hours if immunosuppressed, if Listeria monocytogenes (adults >50years) is suspected (child 60mg/kg to 2,4g)

- Penicillin/Cephalosporin anaphylaxis: Chloramphenicol 25mg/kg IV

Below list has recommendations for therapy after pathogen identification by positive Gram stain. Multiple studies have shown an association between time of antibiotic administration and poor outcome, is greater above 4-6 hours.

- S. pneumonie = Ceftriaxon + Vancomycin
- N. meningitidis = Ceftriaxone
- Listeria monocytogenes = Ampicillin or penicillin G
- S. agalacticae = Ampicillin or penicillin G
- H. influenzae = Ceftriaxone

While controversial, adjunctive steroids are recommended in all suspected cases of bacterial meningitis especially with Streptococcus pneumoniae meningitis and should be given before or with the first dose of antibiotic. Give

Dexamethasone, 0,15 mg/kg (max. 10 mg) IV every 6 hours for 4 days in adults and 0,4 mg/kg IV every 12 hours for 2 days for children. There is no evidence of mortality benefits but prevention of hearing loss and long-term neurologic squeals.

As we are treating meningitis in the emergency department, it is reasonable to give a dose of dexamethasone with the first round of antibiotics for the agent is unknown.

Primary treatment of viral meningoencephalitis is symptomatic. In meningitis caused by HSV-1 or 2, or severe EBV and VZV Acyclovir is added in therapy. Seriously ill patients should receive Acyclovir IV 15-30 mg/kg per day in 3 divided doses, which can be followed by an oral dose, 800 mg, five times daily for the total course of 7-14 days. Patients who are less ill can be treated with oral drug alone.



## Prevention and prophylaxis

Patients hospitalized with suspected *N. meningitidis* infection or meningitis of uncertain etiology require droplet precautions for the first 24 hours of treatment or until *N. meningitidis* can be ruled out.

Those who came in close contacts with an infected person, especially with *N. meningitidis* or *H. influenzae*, give Rifampin (600mg/12h oral for 2 days; children >1year 10 mg/kg/12h, <1year 5 mg/kg/12h) or Ciprofloxacin (500mg oral, 1 dose 250mg for child 5-12 years). In pregnancy give a single dose of Ceftriaxone 250mg IM or ciprofloxacin 500 mg oral.

## Procedures

If there is suspicion of bacterial meningitis, the emergency physician should perform tasks in the following order: blood cultures, steroids, antibiotics, CT and LP

## Pediatric, Geriatric and Pregnant Patients

An atypical presentation is common in elderly (>65 years) as lethargy, the absence of fever and minimal signs of meningismus. Older adults and people with additional medical conditions may only present with a slight headache and fever or general weakness.

Neonates, infants and young children usually show poor feeding, irritability, and fever. In babies, a fever, irritability, decreased appetite, rash, vomiting, and a shrill cry may point to meningitis. Other signs include stiff body and bulging soft spots on the head that aren't caused by crying. Babies with meningitis may cry when handled.

Young children with meningitis may have flu-like symptoms, cough or respiratory distress. In children, history of respiratory tract infection is common, and they are also more likely than adults to experience a seizure. When a child is looking sick

and has a fever, has bacterial meningitis until proven otherwise.

Laboratory findings in blood and CSF can be normal in extreme ages.

## Disposition Decisions

Admission criteria: If there is clinical suspicion of meningitis, patients should be admitted for further workup and treatment.

ICU Referral: Patients with signs of shock or septicemia must be admitted to Intensive unit care (ICU). These signs include capillary refill time more than 4 seconds, unusual skin colour or rapidly progressive rash, systolic hypotension <90mmHg, pulse rate <40 or >140/min, respiratory rate <8 or >30/min, acidosis pH < 7,3 or base excess more negative than -5, white blood count < 4×10<sup>9</sup>/L, lactate > 4mmol/L, GCS < 12 or a drop of 2 points, moribund state, altered mental state/decreased conscious level, poor urine output, poor response to initial fluid resuscitation.

References and Further Reading, click [here](#)

# Sinusitis

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by Katja Žalman and Gregor Prosen

## Case Presentation

*The 32-year-old married woman presented with nasal stuffiness with yellow nasal drainage, pain over the cheek, obstructed nose, facial pain and pressure, subjective fever and chills, mildly productive cough and overall malaise for ten days. She has used over-the-counter medication without significant benefit. She smoke three packs of cigarettes per week. She takes no medications and denies chronic medical diseases.*

*She is conscious (GCS 15), alert and oriented. BP 125/78, HR 77/min, RR 19 breaths/min, body temperature 38 °C. HEENT exam shows intact external ocular muscles, pupils are equal, round and reactive to light, red swollen nasal mucosa with thick yellow-green discharge. No polyps noted. Right maxillary and frontal sinuses are tender to palpation. Mild erythema noted on posterior oropharynx. External ear canals have no*

*edema or erythema. The tympanic membranes are neither bulging or retracted; the ear landmarks are easily identifiable. The neck is supple without lymphadenopathy. The chest is clear to auscultation bilaterally.*

## Introduction

Sinusitis is one of the most common infections treated by emergency physicians and affects about 1 in 8 adults in the north America. It is the fifth most common diagnosis for which antibiotics are prescribed.

Sinusitis is an inflammation of the paranasal sinuses (frontal, maxillary, ethmoid and sphenoid).

A healthy sinus is sterile and lined with a thin layer of mucus that traps dust, germs and other particles in the air. Tiny, hair-like projections in the sinuses seep the mucus towards ostial opening that leads to the back of the throat, and then they slide down to the stomach.

Sinusitis rarely occurs without concurrent rhinitis and inflammation of the contiguous, nasal mucosa is simultaneously involved, and therefore the preferred term for this condition is rhinosinusitis.

Rhinosinusitis is classified according to the new guidelines (2015) by duration as;

- acute rhinosinusitis (ARS) – when the illness appears less than 4 week
- chronic rhinosinusitis (CRS) when lasting more than 12 weeks, with or without acute exacerbations. Chronic rhinosinusitis should be confirmed as the clinical diagnosis. Objective



documentation of sinonasal inflammation may be achieved using anterior rhinoscopy, nasal endoscopy or computed tomography.

The different subgroups of acute rhinosinusitis are based on the duration of symptoms and signs, into acute bacterial rhinosinusitis (ABRS) or viral rhinosinusitis (VRS). Four or more episodes of rhinosinusitis per year, without persistent symptoms in between, the state is termed as recurrent ARS.

The acute rhinosinusitis is most frequently (90%) associated with viral upper respiratory tract infection. It is the most important risk factor for the development of acute bacterial sinusitis and it is most often caused by rhinovirus, coronavirus, influenza A and B, parainfluenza, respiratory syncytial virus, adenovirus, and enterovirus.

The most common occlusions that leads to bacterial overgrowth and excess mucus production are allergies, trauma and fractures, mechanical obstruction

from tumors, abnormal anatomy, weaker immune system, nasal polyps and also nasogastric and nasotracheal intubation. The primary pathogens responsible for acute bacterial and recurrent ARS are *Streptococcus pneumoniae*, non-typable *H. influenzae*, and *M. catarrhalis*.

In chronic sinusitis, however, anaerobic bacteria, streptococcal species, *S. aureus* and also fungi (*Rhizopus*, *Aspergillus*, *Candida*, *Histoplasma*, *Blastomyces*, *Coccidioides*, and *Cryptococcus* species) play role.

## Critical Bedside Actions and General Approach

First, check conditions and vital signs of the patient, and stabilize them if necessary. The most of the patients are rarely need any intervention during the primary evaluation (ABC) stage. When the patient is stable, we can continue with taking the history and physical exam, list of differential diagnoses, general diagnostic and appropriate treatments. The acute care of a patient diagnosed

with acute rhinosinusitis is to eradicate infection, decrease severity and duration of symptoms and prevent complications.

## Differential Diagnosis

The diagnosis of rhinosinusitis consists of the combination of clinical history, physical examination, imaging studies, and laboratory tests.

## Conditions that predispose to rhinosinusitis are

- Allergic and nonallergic rhinitis
- Anatomic abnormality of the ostiomeatal complex
- Aspirin sensitivity
- Associated conditions: asthma, otitis media
- Churg Strauss syndrome
- Ciliary dyskinesia,
- Cocaine abuse
- Cystic fibrosis

- GERD
- Immune diseases and immunocompromised status
- Instrumentation (nasogastric and nasotracheal intubation)
- Kartagener syndrome,
- Nasal anatomic variants
- Nasal polyps
- Rhinitis medicamentosa
- Trauma
- Tumors
- Young syndrome

## Differential diagnosis of rhinosinusitis

- Allergic rhinitis (seasonal, perennial)
- Anatomic abnormalities (foreign body, nasal polyps, nasal septal deviation, enlarged tonsils and adenoids)
- Cerebral spinal fluid rhinorrhea

- Concha bullosa and other middle turbinate abnormalities
- Infectious rhinitis (viral upper tract infections)
- Nonallergic rhinitis (vasomotor rhinitis, aspirin tolerance, eosinophilic nonallergic rhinitis)
- Rhinitis medicamentosa (decongestants,  $\beta$  – blockers, birth control pills, antihypertensives)
- Rhinitis secondary to: pregnancy, hypothyroidism, horner sindrom, weger granulomatosis – midline granuloma
- Tumors
- Vascular headache (migraine)

## History and Physical Examination Hints

Before we start the focused physical exam, which is based on an examination of the respiratory system, we have to take a look at a patient's history and have to be especially focused on:

- signs and symptoms (major and minor)
- questions on allergic symptoms (sneezing, watery rhinorrhea, nasal itching and itchy watery), asthma and immunocompromising disorders
- history of previous episodes of rhinosinusitis
- history or possibility of trauma, fractures, nasal anatomic variants and anatomic abnormality of the ostiomeatal complex
- active or passive smoking
- current medications

Symptoms associated with rhinosinusitis are divided into major and minor groups. Combinations of these symptoms provide a diagnosis based on the patient's history, viewed by anterior rhinoscopy, or as a postnasal discharge on pharyngeal examination.

## Major Symptoms

- Facial pain/pressure/fullness
- Fever (for acute sinusitis only)
- Hyposmia/anosmia
- Nasal obstruction/blockage
- Nasal or postnasal discharge/purulence

### Minor Symptoms

- Cough
- Dental pain
- Ear pain/pressure/fullness
- Fatigue
- Fever (for subacute or chronic sinusitis)
- Halitosis
- Headaches

Acute rhinosinusitis is diagnosed when a patient presents with up to 4 weeks of purulent nasal drainage, nasal obstruction, facial pain-pressure-fullness, or all of these symptoms. When a patient meets the criteria for ARS, the clinician

should distinguish between viral rhinosinusitis (VRS) and bacterial ABRs. It can be difficult to distinguish between acute viral from acute bacterial sinusitis.

P – Facial **pain**, pressure or fullness (may involve the anterior face, periorbital region or manifest with headache)

O – Nasal **obstruction** (congestion, blockage, stuffiness)

D – Nasal purulence or discolored postnasal **discharge** (infected, colored, oozing)

S – Hyposmia or anosmia (**smell**)

Acute rhinosinusitis typically progresses over a period of 7 to 10 days; it is mostly self-limited and resolves spontaneously.

During a viral upper respiratory tract infection, three common clinical presentations should guide the clinician to think that it is an episode of acute bacterial sinusitis: persistent symptoms, severe symptoms, or worsening symptoms.

•When symptoms or signs (PODS) – the presence of  $\geq 2$  PODS symptoms, one of which must be O or D of ARS lasting for more than 10 days but less than 30 days without any evidence of clinical improvement.

- Onset with severe symptoms or signs of high fever ( $\geq 39^{\circ}\text{C}$  [ $102^{\circ}\text{F}$ ]) and purulent (infected, colored or oozing) nasal discharge or facial pain lasting for at least 3–4 consecutive days at the beginning of the illness.
- Onset with worsening symptoms or signs characterized by the new fever (fever is present in some patients with VRS in the first few days of illness but does not predict bacterial infection as an isolated diagnostic criterion – it has a sensitivity and specificity of only about 50% for ABRs), headache, dental pain, or increase in nasal discharge, following a typical viral upper respiratory infection (URI) that lasted 5–6 days and were initially improving (“double-worsening”).

Chronic rhinosinusitis is diagnosed when a patient presented greater than 12 weeks of anterior or posterior mucopurulent drainage, nasal obstruction, facial-pain-pressure-fullness and decreased the sense of smell.

Invasive fungal sinusitis usually occurs in immunocompromised patients and patients with diabetes. It is generally associated with fever, nasal pain, cloudy rhinorrhea, and affected turbinates by dark, thick and greasy material.

The anterior rhinoscopic examination is best performed after the application of a topical decongestant. The status of the nasal mucosa, the presence and color of nasal discharge should be evaluated. Predisposing anatomical variations can also be noted during anterior rhinoscopy.

The endoscopic examination should be used in selected patients with chronic or recurrent sinusitis, in the patient with rhinosinusitis who do not respond to therapy as expected, and in younger children in whom a medical history is

deemed unreliable. Endoscopy provides ideal direct visualization of the nasal cavity, and anatomical structures such as Eustachian tube orifice, tonsils, posterior tongue, epiglottis, glottis, and vocal cords. The nasal polyps can be identified, as well as the presence of purulent ostial secretions. Endoscopy is usually performed by otolaryngologists. Therefore, emergency physicians should chose the patients who needs proper referral.

## Emergency Diagnostic Tests and Interpretation

### Imaging

In the majority of patients with rhinosinusitis, radiographic imaging is unnecessary in case of meeting diagnostic criteria for acute rhinosinusitis. Imaging procedures are useful when symptoms are vague, in poor response to initial management, comorbidities that predispose complications, atypical presentation and a history of trauma.

Ultrasound is safe, rapid and noninvasive for evaluating only the maxillary and frontal sinuses. The A-mode may be useful for screening the fluid in the maxillary sinus, and the B-mode detecting fluid in the cavity, mucosal thickening, or soft tissue mass in the maxillary sinus.

X-rays is not recommended for patients who have already met the clinical diagnostic criteria for ABRS. Radiography cannot be used to distinguish between bacterial and viral etiologies.

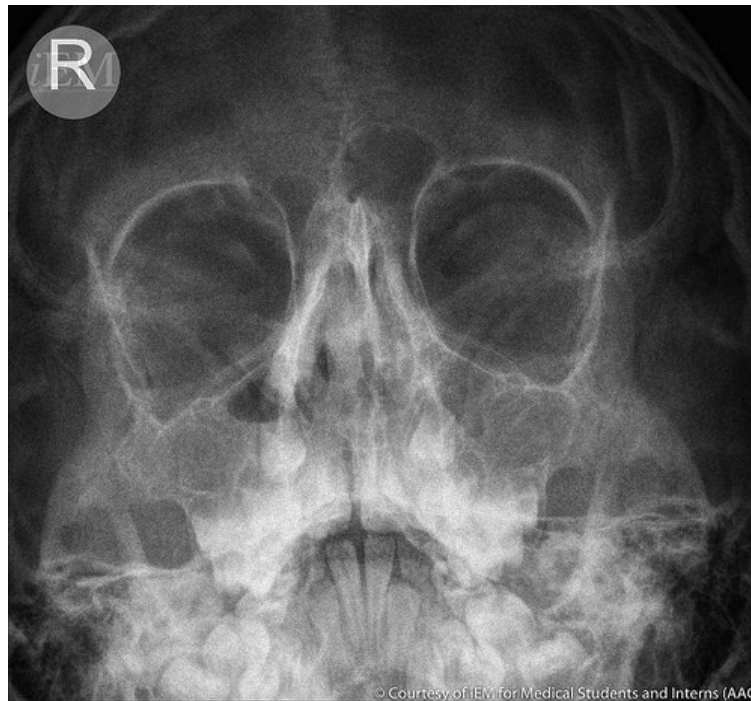
### X-rays includes 3 different projections:

- Waters view (occipitofrontal) – for maxillary and frontal sinuses
- Caldwell view (angled posteroanterior) – only that visualizes the ethmoid air cells
- Lateral view – visualize the sphenoid sinus and primary for adenoids in children



## Radiographic findings of acute sinusitis are;

**Image 13.2**

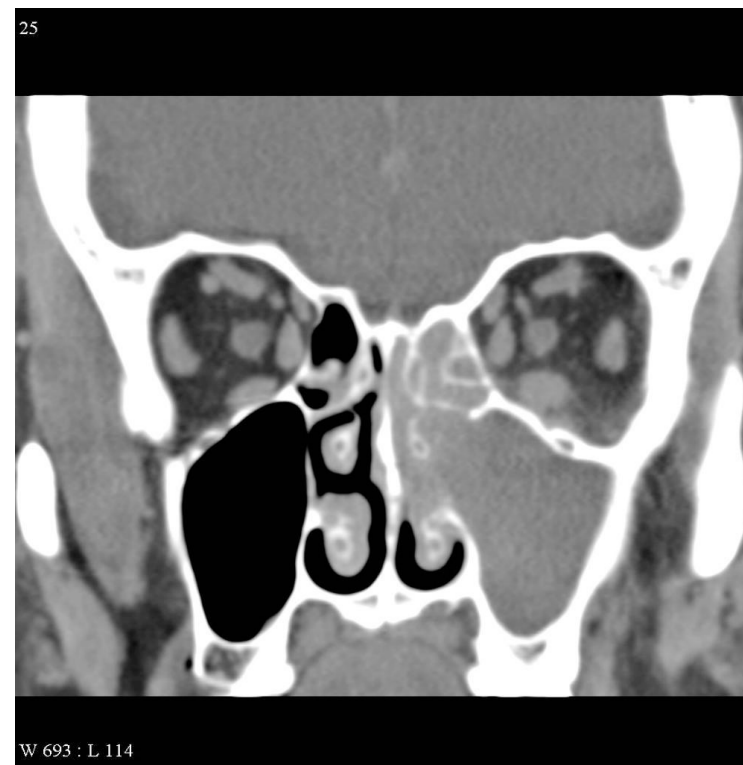


*X-ray – The Waters view shows air fluid level on the right maxillary sinus, and loss of air on the left maxillary sinus. Air presence is also decreased in ethmoidal sinuses.*

- Diffuse opacification,
- Mucosal thickening (>4 mm), or an air-fluid level.
- Mild-to-moderate mucosal thickening, however, is a nonspecific finding.

CT is not used for routine evaluation and is limited to chronic and recurrent sinusitis, causes of questionable diagnosis, patients with unresponsive disease, immunocompromised patients with fever, dentomaxillary pain or investigation of complications (severe headache, facial swelling, cranial nerve

**Image 13.3**



*Case courtesy of A.Prof Frank Gaillard, Radiopaedia.org. From the case rID: 4890*

palsies, or forward displacement or bulging of the eye – proptosis). More than

50% of patients with a recent upper respiratory infection have abnormal findings on CT scan. On the CT with

**Image 13.4**



*Case courtesy of Dr Bruno Di Muzio, Radiopaedia.org. From the case rID: 31870*

acute rhinosinusitis, we can find opacification, air-fluid level, and severe mucosal thickening.

MRI is not used for routine evaluation. MRI is a sensitive technique for evaluating suspected fungal sinusitis and

for differentiating between inflammatory disease and malignancy.

### Laboratory Tests

Complete blood cell count (CBC) is generally not specific, and it is unnecessary for the majority of patients with uncomplicated rhinosinusitis. In most cases, the results show that the CBC may be within normal ranges.

Higher level of erythrocyte sedimentation and C-reactive protein level can be seen in patients. Both of them are not specific.

Nasal cytology can be useful with variety of syndromes, including allergic rhinitis, bacterial sinusitis, eosinophilia, nasal polyposos, and aspirin sensitivity.

The culture of secretions from the nasal cavity or nasopharynx do not differentiate ABRS from VRS and are not routinely obtained unless in immunocompromised, intensive care patients and patients with complications of rhinosinusitis.

## Emergency Treatment Options

### Viral rhinosinusitis (VRS) treatment

Viral rhinosinusitis is a self-limited disease that occurs from 2 to 5 times per year in the average adult. Decongestant therapy such as topical steroids, topical and/or oral decongestants, which can not be used more than 3 to 5 day, mucolytics, and intranasal saline spray. They may be used alone or in varying combinations. Analgesics or antipyretic drugs (acetaminophen, ibuprofen, or other nonsteroidal anti-inflammatory agents) may be given for pain and fever.

### Bacterial rhinosinusitis (BRS) treatment

Delaying antibiotic treatment of ABRS for up to 7 days after diagnosis is the current approach. This allows the infection get better on its own. If not, prescribe initial antibiotic therapy for adults with uncomplicated ABRS.

The clinician must also consider the patient's age, general health, cardiopulmonary status, and comorbid conditions when assessing suitability for watchful waiting.

When we decide to treat ABRS with an antibiotic, the commonly used drug for children and adults is amoxicillin (with or without clavulanate as first-line therapy). A period from 5 to 10 days regimen of amoxicillin 500 mg, 2 times a day is recommended by many as the first-line therapy. The acute sinusitis generally responds to treatment from 10 to 14 days. Some physicians continue treatment for 7 days after the patient is well to ensure complete eradication of the organism and prevent relapse.

For patients who do not respond to amoxicillin, allergic to or intolerant of amoxicillin, live in communities with a high incidence of resistant organisms, failure to respond within 48-72 hours, persistence of symptoms beyond 10-14 day the second-line therapy is the most

commonly used, which include cephalosporins, macrolides or quinolones.

Adjunct therapy such as intranasal saline irrigations, intranasal corticosteroids and local topic decongestants (oxymetazoline hydrochloride) is recommended. Topical agents should be used for up to 5 days; as extended use results in rebound vasodilation and nasal obstruction, the condition is termed as “rhinitis medicamentosa.” Antihistamines are not recommended as adjunct therapy unless there are patients with a history of allergic rhinosinusitis.

In patients with complications of acute rhinosinusitis high-dose intravenous antibiotics, including cefuroxime, ceftriaxone, or ampicillin-sulbactam is recommended.

## **Pediatric, Geriatric, Pregnant Patient, and Other Considerations**

The described procedures can also be used in the pediatric, geriatric and pregnant patient with rhinosinusitis.

## **Disposition Decisions**

Patients with uncomplicated rhinosinusitis can be discharged home with prescription for decongestant therapy, nonsteroidal anti-inflammatory drugs and in the case of ABRH with appropriate antibiotics. All other patients with complications require additional work-up or admission.

**References and Further Reading**, click [here](#)

# Sepsis

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by Emilie J. Calvello Hynes

## Case Presentation

*74 y/o female with history of diabetes, hypertension and coronary stent placement presents with confusion and cough. She has had a cough for 2 days and saw her primary care doctor who prescribed an antibiotic. Her husband describes her as behaving normally until today 3 hours prior to presentation. She is taking Insulin, Lisinopril, Aspirin, Metoprolol, and Azithromycin. There are no allergies to medications.*

*On exam, the patient is spontaneously breathing with eyes closed but opens to verbal command. She is lethargic and oriented only to self. Blood pressure is 92/48mmHg, heart rate 122/min, respiratory rate 24 cycles/min, temperature is 37.5 °C and oxygen saturation is 89% on room air. She has dry mucous membranes, and her skin is cool and diaphoretic. The lung exam reveals crepitations in the left base and heart*



*sounds are regular but tachycardic. Her abdomen is flat and non-tender, and her neurological exam reveals no focal deficits.*

## Introduction and Definitions

In the last 20 years, the collective understanding of sepsis care has gone through a major transformation. The term sepsis describes a physiologic syndrome with characteristic biochemical abnormalities initiated by infection. While the mortality from sepsis in many high-income countries is decreasing, the reported incidence has found to be increasing due to aging populations as well as greater attention paid to early recognition of a potentially deadly syndrome. The true incidence of sepsis worldwide is unknown, and there is no doubt, even with the most conservative estimates, that sepsis is a leading cause of critical illness and death on the planet.

Sepsis recognition and appropriate care have a huge possible impact on human mortality, and yet the public health awareness of sepsis is quite poor. Sepsis can have many presentations making it sometimes difficult for even the most experienced physicians to detect, further emphasizing the need for clear definitions



to prompt the right treatment interventions.

Prior definitions of sepsis were predicated on the inflammatory response from the host, termed the systemic inflammatory response syndrome (SIRS).

## Systemic Inflammatory Response Syndrome

### Two or more of the following

- Heart rate > 90 beats/min
- Respiratory rate > 20 cycles/min or PaCO<sub>2</sub> <32 mm Hg
- Temperature > 38°C or < 36°C
- WBC > 12,000/mm<sup>3</sup>, < 6,000/mm<sup>3</sup> or > 10% bandemia

SIRS can be caused by a large variety of inciting agents that induce host inflammatory response, such as burns, pancreatitis, a variety of infectious organisms and toxins. Sepsis, for the last two decades, has been understood to be

SIRS plus an infectious source. Severe sepsis was defined as sepsis plus organ dysfunction. Whereas, septic shock was defined as hypotension induced by sepsis that persisted despite adequate fluid resuscitation. However, a recent task force has fundamentally shifted the previously understood definitions of sepsis in the following important ways. (Table 13.3)

SIRS criteria is not adequately sensitive or specific in identifying those who may go on to have significant morbidity and mortality from overwhelming infection. Rather, sepsis is now defined as life-threatening organ dysfunction due to a dysregulated host response to infection. Organ dysfunction is best defined in the undifferentiated emergency department patient by the quickSOFA score (qSOFA). [SOFA = Sepsis related Organ Failure Assessment Score]

Identification of these patients should prompt further diagnostic evaluation for end-organ damage. The definition of

**Table 13.3** Comparison of Sepsis Definition Evolution

OLD DEFINITION (PRE 2016)	CURRENT DEFINITION
Sepsis = SIRS + infectious source	Sepsis = life-threatening organ dysfunction due to a dysregulated host response to infection <ul style="list-style-type: none"> <li>• ED patients: 2 or more of the following qSOFA score may identify patients with increased mortality</li> <li>• SBP less than or equal to 100 mm Hg</li> <li>• RR <math>\geq</math> 22</li> <li>• Altered mental status (GCS <math>&lt;</math>15)</li> </ul>
Severe sepsis = sepsis + organ system dysfunction	The term severe sepsis is NO LONGER USED.
Septic shock Adults = persistent arterial hypotension despite adequate fluid resuscitation Pediatrics* = tachycardia with decreased signs of perfusion	Septic Shock = subset of patients with sepsis and profound circulatory, cellular, and metabolic abnormalities  Clinical Criteria Despite adequate volume resuscitation, Persistent hypotension requiring vasopressors to maintain MAP $\geq$ 65 mm Hg Lactate $\geq$ 2 mmol/L

*Original by the author*

*Read for new definitions from - Rubulotta FM, et al. Crit Care Med. 2009;37(1): 167-170.*

*\* Because of higher vascular tone, neonates and children may be in a shock state long before manifestation of hypotension.*

septic shock has been simplified as a subset of sepsis in which underlying

circulatory and cellular/metabolic abnormalities are profound enough to increase mortality substantially. These patients are identified by persistent hypotension and having lactate greater than 2 mmol/L after fluid resuscitation. Of note, the term severe sepsis should no longer be used as all sepsis is considered to have a high probability of being severe.

## Critical Bedside Actions and General Approach

For the critically ill patient, you must make a rapid determination of syndromic category of their acute illness. If they meet the above definition of sepsis and there is a concern for ongoing shock, proceed to the critical bedside actions.

### First 5 – 10 minutes if high suspicion for septic shock

- Transfer to critical care room if available
- Vital signs, primary bedside evaluation with ABC..., administer O<sub>2</sub>, attach to the cardiac monitor
- 2 large bore IVs

- Check glucose
- Start 2 L IV Fluid bolus (LR or NS) for adults or 20 mL/kg for pediatrics (unless malnourished)
- Be prepared to assist with airway patency or protection (i.e., intubation) if necessary
- Prepare broad-spectrum antibiotics for administration within the first hour

## Differential Diagnosis

### Differential Diagnosis of Sepsis – non infectious etiologies

#### Shock states

- Cardiogenic shock
- Hypovolemic shock
- Hemorrhagic shock
- Obstructive shock
- Distributive shock

#### Cardiac/pulmonary

- Myocardial infarction

- Congestive heart failure

- Pulmonary embolism
- Acute respiratory distress syndrome

#### Environmental

- Heat stroke
- Burns

#### Endocrine

- DKA
- Adrenal crisis
- Thyrotoxicosis
- Pancreatitis
- Hypoglycemia

#### Toxicologic

- Salicylate toxicity
- Neuroleptic Malignant Syndrome
- Serotonin syndrome
- Sympathomimetic toxidrome
- Delirium tremens

## Neurologic

- Status epilepticus
- Cerebral hemorrhage

## History and Physical Exam Hints

### History

- History of immunocompromise (HIV, chemotherapy, etc.), alcoholism, malignancy, liver disease, diabetes, ongoing steroid use, intravenous drug use
- Travel history, vaccination status
- Recent illness, sick contacts
- History associated with source: cough, dysuria, shortness of breath, chest pain, abdominal pain, vomiting, diarrhea, back pain, decreased urine output, focal neurological deficits, rash or skin changes, change in mental status
- Pediatric-specific: increased work of breathing, decreased PO intake, change in behavior

**Table 13.4** Findings Associated with an Infectious Source

FINDING	SOURCE
Pulmonary findings	Pneumonia, empyema, parapneumonic effusion
Urine appearance	URI, pyelonephritis, infected renal calculi
Skin findings (wounds, rash, crepitus, bullae)	Cellulitis, abscess, meningococcus, viral or tick borne disease, gangrene, necrotizing fasciitis
Focal neurologic deficit	Cerebral abscess, epidural abscess
Bony findings (pain or asymmetry in extremities)	Myositis, discitis, osteomyelitis, septic joint
Peritoneal signs	Intra-abdominal abscess/ inflammation, perforation, spontaneous bacterial peritonitis
Heart findings (rub, murmur)	Pericarditis, endocarditis
Stridor	Epiglottitis, tracheitis, croup
Vaginal discharge	Pelvic inflammatory disease, endometritis, septic abortion, chorioamnionitis

*original by the author*

## Physical Exam Signs

- Vital Signs: Tachycardia, fever, low BP, tachypnea
- Poor perfusion: hot or cool skin, altered mental status, poor urine output ( $<0.5$  mL/kg/hr), weak pulses,
- Pediatric-specific: skin mottling, delayed capillary refill skin, poor urine output ( $<1$  mL/kg/hr)
- Associated finding less likely to be non-infectious source: chest pain, evidence of DVT, evidence of ingestion (pill fragments)

## Emergency Diagnostic Tests and Interpretation

### Labs

- Specific derangements in sepsis: creatinine, LFTs, bilirubin, platelets, coagulation studies
- Serial serum lactates



- Source testing (guided by H&P): UA, CSF, pleural, intraperitoneal, synovial fluid
- Cultures: Blood, urine
- CSF, body fluid if indicated
- Cultures are positive in sepsis only 30-40% of the time
- Special tests (if indicated): malaria, dengue, viral hemorrhagic fever, etc.

## Imaging

- Guided by history and physical
- Chest x-ray for all
- Bedside ultrasound of IVC to monitor resuscitation

**Video tutorial** (1 and 2) for IVC measurement

Consider based on history:

- abdominal CT,
- head CT,

- spine MRI,
- extremity X-ray

## Emergency Treatment Options

Mortality for sepsis and septic shock can be as high as 40-50%. Decreases in mortality are accomplished via two goals:

1. Restore tissue perfusion
2. Locate and treat infectious source

## Restore tissue perfusion

### Fluids

Resuscitation with normal saline

- General recommendations for adults include 20-40 mL/kg in defined volumes amounts (500 mL) with reevaluation after every bolus administration.
- Pediatrics – start with 20 mL/kg bolus
  - Significant caution in those malnourished or severely anemic

Hydroxyethyl starch should be avoided.

Albumin may be used if indication AFTER adequate crystalloid resuscitation.

Monitor for signs of fluid overload (increasing hypoxia, rales, hepatomegaly in children).

### Oxygenation

Target saturation of > 90%

Intubation for respiratory failure and consider for those in refractory septic shock.

### Vasopressors

Provide for those with MAP < 65 mmHg despite adequate fluid resuscitation. (MAP = [(2 x diastolic) + systolic]/ 3)

Initial vasopressors may be given peripherally initially, although central venous access preferred.

Norepinephrine (0.01-3 mcg/kg/min) the preferred choice, with the recommended second agent of epinephrine (0.1 – 1 mcg/kg/min) or vasopressin (0.03 units/min)

## Steroids

Consider for those with MAP < 65 mmHg despite fluid and vasopressor therapy.

Hydrocortisone (adults – 200 mg, pediatric 1-2 mg/kg) or equivalent

## Locate and treat infectious source

### Location

- Guided by history and physical
- Source control:
  - Debride or drain any localized source of infection; surgical consult for deeper infections such as intrabdominal abscess or empyema.

- Remove catheters and lines associated with infection.

### Timing

Critically ill patients should have antibiotics given within 1 hour.

Do not delay antibiotics for testing!

### Antibiotics

Choice of antibiotics driven by local resistance patterns, region-specific epidemiology (HIV, malaria, influenza, etc. prevalence), and availability of drugs

Those with septic shock should always include broad-spectrum coverage (gram positive, gram negative, anaerobes).

Consider antimalarials where appropriate.

Consider specific anti-viral therapy when appropriate (i.e., acyclovir for meningoencephalitis, oseltamivir for influenza in the immunocompromised host).

## Pediatric, Geriatric, Pregnant Patient and Other Considerations

### Pediatric

Neonates and immunocompromised (sickle cell, oncology, diabetic and HIV) patients are at particular risk for overwhelming infection.

Controversy exists regarding fluid management in developing countries with high malaria prevalence rates with fluid boluses found to increase mortality.

For respiratory distress and hypoxemia, make early use of high flow nasal cannula and CPAP while starting resuscitation.

Extracorporeal Membrane Oxygenation (ECMO) may be considered for refractory pediatric shock and respiratory failure.

### Geriatric

Increased risk factors due to comorbidities, endocrine deficiencies, pre-existing malnutrition, and age-related immunosenescence.

Diagnosis may be more difficult as the initial inflammatory response to infection may be blunted or absent.

1.5 higher mortality rates than younger patients.

## **Pregnant**

The etiology of sepsis in pregnant women is expanded and includes septic abortion, chorioamnionitis/endometritis, group A Streptococcus infection, particular susceptibility to influenza and necrotizing vulvitis.

Adequate resuscitation of the mother often will improve outcomes for the fetus; however, in the critically ill patient, early delivery of the fetus should be considered with appropriate consultation.

## **Disposition Decisions**

All patients with suspected sepsis should be admitted.

ICU level care for those who meet criteria for septic shock.

**References and Further Reading**, click [here](#)

# Selected Toxicologic Problems





# Opioid Overdose

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by Aldo Emigdio Bartolini Salinas and Jesús Daniel López Tapia

## Case Presentation

*A 22-year-old male was brought to the emergency room by EMS at 7 pm. His parents arrived at the hospital and mentioned that this was not the first time their son had a similar event. The patient was lethargic upon his arrival so clinical history was difficult to obtain from the patient and the parents had no additional information to provide.*

*On physical examination, the patient was lethargic and slightly bradycardic with 59 bpm, his respiratory rate was 14 per minute. Body temperature was stable at 36.6°C and blood pressure was 105/80 mmHg. Oxygen saturation at room air was 94%. Bilateral pupils were miotic, but no clear measurement was obtained. On auscultation, cardiac and lung sounds were rhythmic and stable. Neurologic examination was accurate. Gastrointestinal auscultation showed hypoactive bowel sounds. When blood pressure was measured there*

*were needle marks on both of his forearms. The rest of the systematic evaluation was normal. Electrocardiogram and laboratory studies were made to rule out other possible diagnoses.*

## **Critical Bedside Actions and General Approach**

In an emergency setting, evaluating circulation, airway, breathing and vital signs are a priority.

Attention must be paid to respiratory rate; pulse oximetry must be done to evaluate oxygen saturation while additional studies are being performed. A ventilation mask with supplementary oxygen to 100% must be given. Eyes, mental status, and skin should be examined. Miotic pupils, euphoria and skin marks are usually found in opioid-intoxicated patients.

A thorough clinical history and physical examination must be done. Further details will be discussed in this chapter.

## **Introduction**

According to the United Nations Office on Drugs and Crime, in 2010 there were more than 15 million opioid consumers worldwide. Opiates are withdrawn from the poppy plant (*Papaver somniferum*), these substances have psychoactive properties and are processed into natural, synthetic and semi-synthetic methods of use. These substances have been used in cultural, recreational

and medicinal settings. Unfortunately, opioid derivatives such as prescription drugs and “recreational” drugs like heroin have been motive of abuse and intoxication, being the number one illicit drug on the market with more deadly outcomes due to abuse.

Opiate pharmacokinetics, toxicology, clinical manifestations, and management will be broadly discussed in this chapter.

## **Epidemiology**

Worldwide opioid abuse, dependence, and intoxication have been increasing in numbers. As of 2015, the United States had an approximate of 2.6 million opioid consumers and addicts. This number includes opioid addicts due to pain relievers and non-prescription opioid consumption. It is vital to consider that heroin use via injection is responsible for the transmission of HIV and hepatitis C. In Russia, Central Asia, and Ukraine, it is responsible for 60-70% of all HIV infections.

Gender distribution of opioid abuse is more common in men than women, ranging in a 3:1 (M: F ratio) for heroin and 1.5:1 for prescription opiates. The most common age of consumers is of 26 years or younger.

## **Pharmacokinetics and Toxicology**

Opioid-related sites in the brain are hypothalamus, thalamus and the limbic system. They bind competitively to an opioid specific receptor. To this date, three specific receptors have been

identified: mu ( $\mu$ ), delta ( $\delta$ ) and kappa ( $\kappa$ ). An opioid-receptor like-1 is still under investigation. These subtypes have a specific and different effect on the body. They are capable of producing cAMP (adenylate cyclase), closing/opening calcium and potassium channels, leading to the ability to hyperpolarize the cell and modulating neurotransmitter release.

There are three action category;

#### Opioid Agonists

- Codeine
- Diphenoxylate-atropine
- Fentanyl
- Heroin
- Hydrocodone
- Loperamide
- Meperidine
- Methadone

#### Opioid Antagonists

- Nalmefene
- Naloxone
- Naltrexone

#### Opioid Agonist-Antagonist

- Buprenorphine
- Nalbuphine
- Pentazocine

Opioids can be consumed orally, by snorting and by subcutaneous or intravenous injection. Its effects vary depending on their site of administration, dose and the type of opioid consumed. If it is taken orally, it may take about six hours to have its maximum effect (methadone) and its clinical effect may persist for 24 to 48 hours. When snorted, its peak effect is usually 30 minutes after. When injected subcutaneously, it may take just about 15 minutes. It may show an immediate effect when done intravenously.

Three main effects caused by opioid consumption are analgesia, euphoria, and anxiolysis. Analgesia occurs by inhibiting transmission from the peripheral nerve to the spinal cord. Anxiolysis happens when opioids act upon noradrenaline releasing neurons located in the locus coeruleus. Euphoria is related to the mesolimbic system dopamine increase. Most opioids are metabolized by the liver to active metabolites and excreted by the kidneys. They have a large distribution volume of 1-10L/kg and are protein-bonded in most cases, which makes hemodialysis a problematic way for opioid clearance.

## History and Physical Examination Hints

Opioids affect the body in various ways. The more consistent clinical effects of opioids are a depressed respiratory rate, changes in mental status, decreased bowel sounds and pupillary constriction (miosis). Additional findings include hypothermia, bradycardia, hyporeflexia, dermal marks.

Pupillary constriction (miosis) is frequent in opioid intoxication. However, normal pupils or mydriasis (pupil dilation) are possible when the patient takes a stimulant simultaneously or when the in case of extended respiratory depression. Therefore, normal pupil examination does not exclude the possibility of intoxication. A thorough and careful examination is a must.

Check respiratory rate to evaluate a suspected opioid-intoxication. A respiratory rate below 12 bpm is a great predictor of toxicity. The pulse oximeter shows oxygen saturation, but a normal reading does not exclude hypercapnia. Monitoring respiratory ventilation via end-tidal CO<sub>2</sub> monitoring and capnography helps to assess diagnosis and possible complications.

Noncardiogenic pulmonary edema is frequent in opioid intoxication. The symptoms include cyanosis, pink bronchial secretions, and rales (with all the additional symptomatology of

intoxication). It is usually resolved once a normal respiratory rate and ventilation are obtained. Cardiovascular changes, mostly bradycardia and hypotension is due to an increase in parasympathetic activity and release of histamine. Lethal ventricular tachyarrhythmias might occur.

Gastrointestinal changes include decreased bowel sounds, peristalsis, and constipation. Additionally, renal changes, particularly renal failure due to rhabdomyolysis may be present (from heroin and methadone abuse). Skin marks due to “skin popping” may be present secondary to subcutaneous injection. Changes in the reproductive system include changes in menstrual cycles, infertility, abnormal prolactin secretion, and decreased libido.

## Differential Diagnoses

Generally, clinical manifestation is enough for diagnosis. When in doubt, laboratory and imaging findings may be helpful. The most common differential diagnoses are the following.

## Drug-induced toxicity

- Ethanol toxicity produces none or little miosis and no gastrointestinal changes. Withdrawal produces hyperthermia and seizures.
- Sedative-Hypnotics toxicity causes less respiratory depression and ataxia in children. Withdrawal produces hyperthermia and seizures.
- Clonidine toxicity causes miosis, hypotension, bradycardia and no gastrointestinal changes.
- Hypoglycemic agents

Organophosphate toxicity causes miosis, vomiting, diarrhea, bradycardia, hypotension or tachycardia and hypertension.

Any medical condition that causes coma.

## Emergency Diagnostic Tests and Interpretation

History and physical examination are generally sufficient to make a diagnosis.



In some complicated cases, laboratory studies, urine screening tests and cardiac screening are recommended.

## Laboratory tests

- Serum or finger-stick glucose measurement (to rule out hypoglycemia)
- Serum acetaminophen concentration (in case of suspected concurrent use with opioids, to rule out suicidal attempt with acetaminophen)
- Serum creatine phosphokinase, blood urea nitrogen, creatinine, urinalysis and serum electrolytes (to rule out rhabdomyolysis).
- Serum ethanol level (to rule out intoxication)
- Blood gas measurement.
- Basic metabolic panel

## Urine toxicologic screenings

Opioids can be detected in urine samples in a maximum period of two days.

Routine urine toxicologic screens are not recommended. They confirm recent abuse, but not acute toxicity.

They can report many false positive results.

## Cardiac screening

Electrocardiographic (ECG) evaluation is strongly recommended in patients who present palpitations, syncope, chest pain and dysrhythmias.

If the initial ECG is normal, a control ECG should be repeated 4-6 hours after.

If the initial ECG shows abnormalities such as QT or QRS prolongation, cardiac monitoring should be done until a normal rhythm.

Some specific cardiac disturbances are presented in the following abused substances:

- Methadone: torsade de pointes, QT prolongation.

• Loperamide: QT or QRS prolongation, ventricular tachycardia.

- Oxycodone: QT prolongation.

## Imaging tests

Plain chest X-rays are reserved for patients who present with symptoms of aspiration pneumonia, respiratory distress syndrome, uncorrected hypoxia or abnormal sounds during lung auscultation.

## Emergency Treatment Options

### Initial Stabilization

In an emergency setting, evaluating circulation, airway, breathing and vital signs are a priority. Ventilatory support should be given with a bag mask and 100% oxygen to patients with respiratory distress or when the proper respiratory function has been jeopardized. If the patient has oxygen saturation above 90% and more than 12 breaths per minute at room air, observation is adequate. However, if oxygen saturation drops

below 90%, supplemental oxygen, bag mask and 0.05 mg intravenous naloxone must be administered to restore normal ventilation. If the blood glucose is 60mg/dl or less, administer glucose intravenously.

### Medications

The first line treatment is Naloxone, a lipophilic, short-acting opioid antagonist, that can effectively reverse opiate-related symptoms. Full patient history is essential to determine whether the patient is a long-term or short-term opioid user, the type of opioid consumed, time of administration and dosage.

The recommended naloxone dose is 0.4 mg for most patients, including those with methadone abuse. It should be diluted in 10 mL of normal saline to reach 0.04mg/ml dilution. Administer 1 ml separate boluses to improve the patients’ respiratory rate above eight breaths per minute. Its clinical effects last up to approximately 70 minutes.

In patients that have abused fentanyl or other synthetic opioids, an increased dosage is recommended. No established dosing is available, but some recommend increasing naloxone dose every 2-3 minutes, starting with 0.5mg -2mg-4mg-10mg and administering a maximum dose of 15mg. Another suggested method of use is by administering naloxone every 2-3 minutes starting with 0.04mg-0.08mg-0.16mg. If respiratory rate is not improved after maximum dose, a different diagnosis must be considered.

### Procedures

Naloxone might be administered intravenously, nebulized and intranasally. A summary of dosage and administration route is discussed in Table 14.1.

**Table 14.1** Naloxone Administration

	INTRAVENOUS NALOXONE	NEBULIZED NALOXONE	INTRANASAL NALOXONE
Recommended Dosage	0.4 mg in 10 mL of normal saline (0.04mg/ml). Administration in separate boluses of 1ml.	2 mg in 3ml normal saline solution with a standard face mask.	1 mg/ml per naris (total dose: 2mg)
Pros.	A gentle method of opioid intoxication reversal, no acute withdrawal symptoms	Easy titration, lower risk of withdrawal symptoms.	Can be used in a patient with complicated intravenous access.
Cons.	Close monitoring is recommended.	No clinical data is available.	It might be difficult to titrate because of unknown absorption rates and bioavailability in humans.

*Adopted and developed from*  
 Li, K., et al (2018). *Annals of Emergency Medicine*, 72(1), 9–11., Stolbach, A., & Hoffman, R. (2018, April 18). *Acute Opioid Intoxication in Adults*. Retrieved from UpToDate: [www.uptodate.com](http://www.uptodate.com), and Yin, S. (2018, January 10). *Opioid Intoxication in Children and Adolescents*. Retrieved from UpToDate: [www.uptodate.com](http://www.uptodate.com)

## Pediatric, Geriatric, Pregnant patient, and other Considerations

### Pediatric population

Morphine and fentanyl might be used for moderate pain in children under 12 years of age and infants, although respiratory depression is frequent. Codeine and tramadol may be used in children older than 12 years old.

The treatment of opioid intoxication is dependent on children's weight. 0.1 mg/kg IV naloxone (max. 2mg per dose) should be administered below 20 kg. 2 mg IV naloxone is recommended over 20 kg.

### Pregnancy

Most opioids are category C and teratogenic, especially in the first trimester. If used for an extended period or during delivery (except for pethidine), opioids may cause respiratory depression in both the mother and the neonate. For chronic pain, opioids should only be given as a last resort. The safer opioids

for pregnant women are codeine, morphine, pethidine, and propoxyphene. In lactation, morphine can be administered, but it must be interrupted every 4 to 6 hours. Buprenorphine and fentanyl are not recommended due to their high concentration in breast milk.

Opioid intoxication during pregnancy can result in severe respiratory distress in both the mother and the neonate. Opioid abuse causes neonatal abstinence syndrome characterized by low birth weight, CNS hyperirritability, myoclonus, hyperreflexia, sweating, vomiting, diarrhea, death, and others. Opioid intoxication during pregnancy requires a neonatologist in the team.

### Geriatric population

Geriatric population may be particularly vulnerable to opioid side effects or toxicity. Lowered doses must and well-established times and days are recommended. Caretakers should be capable and adequately knowledgeable about medication administration. In case

of toxicity or abuse, care must be done by a geriatrician or addictionologist.

## Respiratory or Hepatic pathology

Opioid use should be avoided. If used, close monitoring is of importance.

## Disposition Decisions

### Admission criteria

- Opioid overdoses with short-acting agents may be treated in the emergency department.
- Opioid overdoses with a long-acting (ex. methadone) agents or combinations must be admitted to the ICU.

### Discharge criteria

- A psychiatric evaluation is recommended once the patients' mental status and respiration rate become normal.
- Naloxone should not be administered 2-3 hours before discharge.

## Referral

In case of intentional opioid overdose (as a suicide attempt or drug addiction), the patient must be referred to a psychiatric evaluation or rehabilitation facility.

**References and Further Reading**, click [here](#)



# Selected Eye Problems



# Eye Trauma

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by Serpil Yaylaci and Kamil Kayayurt

## Introduction

The most common cause of unilateral blindness in industrialized countries is eye trauma, most of which can be prevented with the use of protective goggles. Every year, more than 55 million people worldwide experience eye traumas, and more than one million suffer total loss of vision. Eye traumas constitute approximately 3% of total emergency department cases; most of these traumas are minor injuries. Major injuries are less frequent; however, the rate of recovery of visual impairments is quite low in these cases. Sixty-five to eighty percent of eye traumas are observed in males, typically 25–44 years of age. About half of eye traumas are the result of occupational accidents, and the use

of protective goggles reduces the number of those injuries by 70%.

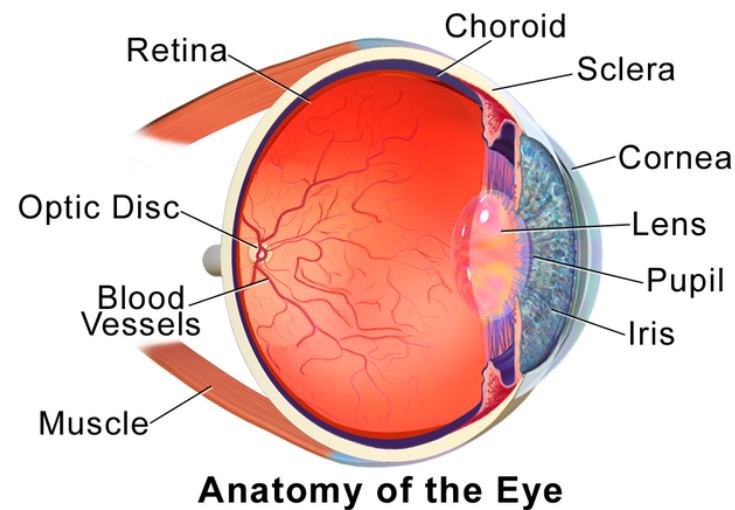
## Anatomy

The eyeball consists primarily of three layers. The outermost layer is a fibrous structure made up of the sclera and the cornea. The middle layer is the vascular layer, called the uvea. The iris, ciliary body, and choroid are located inside the uvea. The innermost layer is the neural layer, called the retina.

The eyeball is located in the bony cavity, called the orbit. The superior wall of the orbit consists of the frontal bone, the lateral wall consists of the zygoma, and the medial and anteromedial walls consist of the maxilla, lacrimal, and ethmoid bones. The [video](#) shows bones of the orbit.

Please visit this [link](#) to see detailed Eye Anatomy videos.

### Illustration 15.1 Anatomy of the eye



Blausen.com staff (2014). "Medical gallery of Blausen Medical 2014". WikiJournal of Medicine 1 (2). DOI:10.15347/wjm/2014.010. ISSN2002-4436

## Orbital Traumas

Eye traumas can be divided into three groups: globe injuries, periorbital injuries, and chemical injuries. Only selected injuries observed most frequently in emergency departments and that can lead to total loss of vision will be discussed.

## Classification of Eye Trauma

### Globe Injuries

#### 1. Open-globe injuries ( Rupture of globe)

#### 2. Closed-globe injuries

- Conjunctival lacerations
- Partial thickness corneal and scleral lacerations
- Corneal and conjunctival abrasions
- Hyphema
- Lens dislocation
- Traumatic iritis
- Retinal detachment
- Vitreous hemorrhage
- Commotio retina

### Periocular trauma

#### 1. Orbita fractures

#### 2. Ekstraocular muscle, vascular, and eyelid injuries

- Orbital compartment syndrome

#### •Eyelid lacerations

- Retrobulbar hematoma
- Traumatic optic neuropathy
- Ophthalmic arter injuries
- Extraocular muscle entrapment

## Globe Injuries

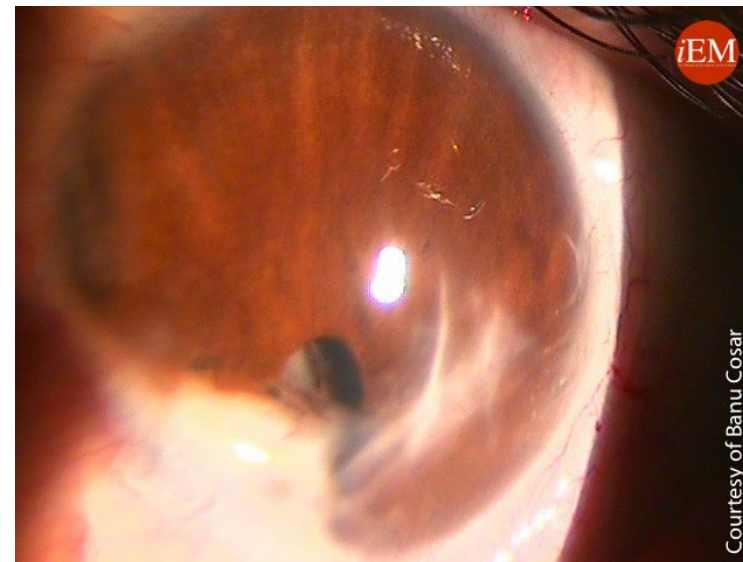
### Globe Rupture

It is an ophthalmologic emergency, consisting of a full-thickness injury in the cornea or sclera caused by penetrating or blunt trauma. Anterior rupture is usually observed, as this is the region where the sclera is the thinnest. Posterior rupture is rare and difficult to diagnose. It can be diagnosed through indirect findings such as contraction in the anterior chamber and decrease in intraocular pressure (IOP) in the affected eye. If there is a risk of globe rupture, a slit lamp test with 10% fluorescein must be conducted. Normal tissue is dark orange under a blue cobalt filter; a lighter color is observed in the damaged zone due to a lower dye



concentration. Ultrasonography (USG) can be useful in making a diagnosis, especially with posterior ruptures. Computed tomography (CT) sensitivity ranges 56–75%. In cases of anterior globe injuries, USG use, and if there is a risk of a foreign metal body, magnetic resonance imaging, are contraindicated. Prompt ophthalmology consultation is required. While in the emergency department, tetanus prophylaxis, analgesics, bed rest, head elevation, and systemic antibiotic therapy are required. The most commonly preferred antibiotics are cefazolin and vancomycin. Age over 60 years; injury sustained by assault, on the street/highway, during a fall, or by gunshot; and posterior injuries are indications of a poor prognosis.

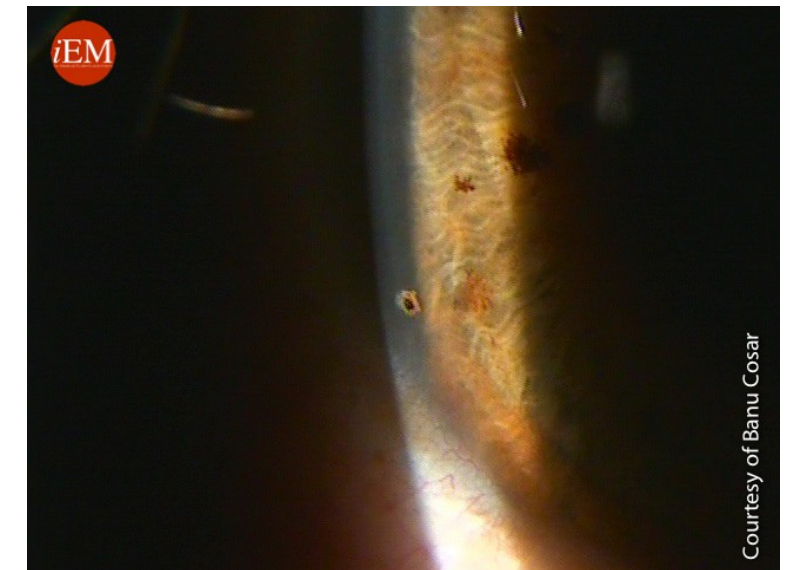
**Image 15.1** ectopic pupil after penetrated eye trauma



## Foreign Bodies

Orbital foreign bodies are classified as superficial or intraorbital. Superficial foreign bodies constitute the second most common general eye injury, after corneal abrasions, and are the most common work-related injuries. They usually consist of earth, stone, wood and metal pieces. Organic foreign bodies have a higher risk of infection. Intraocular foreign bodies (IOFB) are most commonly observed in young males in the form of hammering injuries. Blast injuries and combat injuries are also frequently observed.

**Image 15.2** corneal foreign body

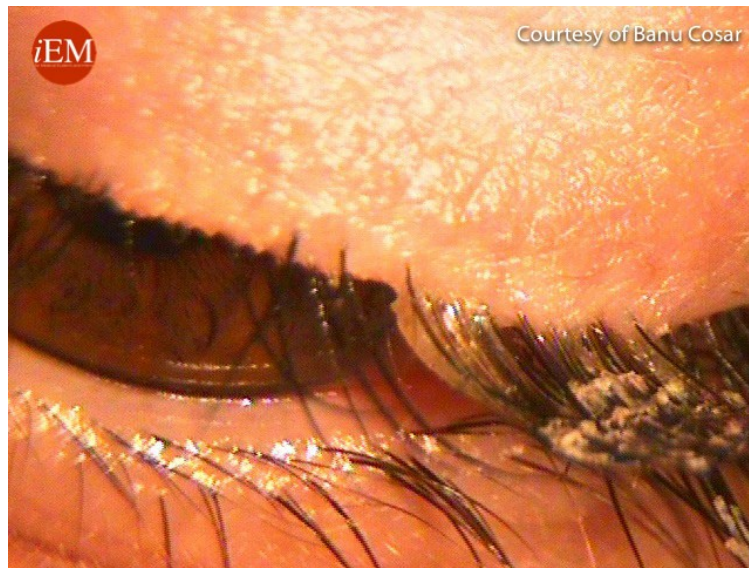


**Image 15.3** corneal foreign body





**Image 15.4** superglued eye



Diagnosis usually depends on patient history. The patient should be asked the location and intensity of the trauma, as well as time elapsed since the injury. In addition, the patient's tetanus risk should be determined. There may be multiple foreign bodies. During the diagnosis, a microscopic examination with fluorescein must be conducted, and it must be determined if there is an intraocular foreign object. After applying local anesthesia, superficial foreign bodies can be removed with saline irrigation or a wet cotton swab. If unsuccessful, an attempt can be made to remove the object with a 25G needle under direct vision using a slit

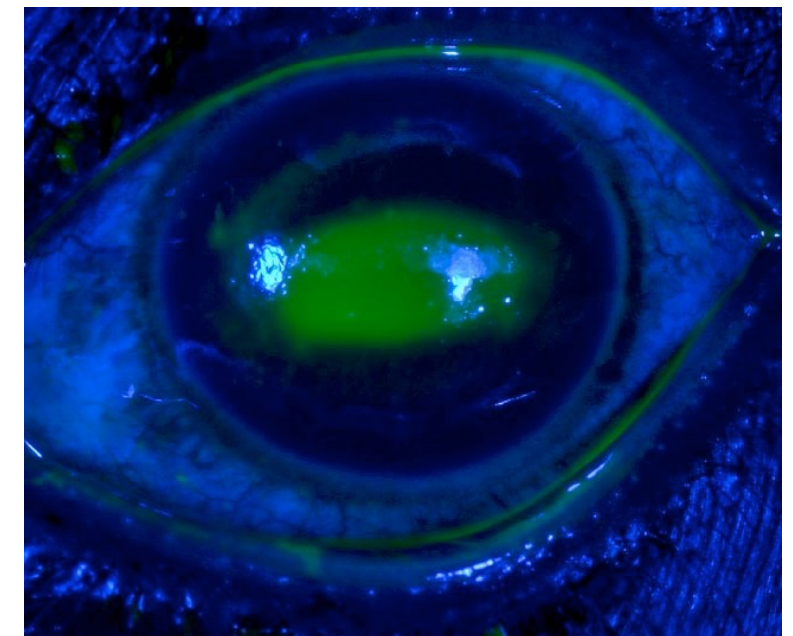
lamp. Any buried foreign bodies should be removed by an ophthalmologist. If there is a risk of IOFB, the patient should be referred to an ophthalmologist. If there is an IOFB, the patient should be treated as a case of globe rupture.

### Corneal Abrasions

These are epithelium defects following a non-penetrating eye trauma; they constitute the most common eye pathology caused by trauma. The most common cause is chronic contact lens use. Other causes include blunt trauma, foreign bodies, burns, and radiation. Symptoms include stinging, burns, pain, and a feeling that there is a foreign object present. During diagnosis, a slit lamp examination must be conducted with fluorescein, and the dimensions and shape of the defect must be ascertained. Linear defects indicate the possibility of a foreign body located in the inner part of the eyelid. Therefore, the inner parts of the eyelid must be examined for foreign bodies as well. In the treatment, preventing bacterial superinfections that

might be caused as a result of the defect, relieving pain, and speeding up recovery must be targeted. Fluoroquinolones are preferred as antibiotics, and oral analgesics are preferred for relieving pain. Topical non-steroids and anesthetics should not be

**Image 15.5** Fluorescein staining confirms the presence of a corneal abrasion



*Courtesy of Simon Arunga. Retrieved from <https://flic.kr/p/NZrhfH>.*

used.

## Hyphema

It is defined as bleeding in the anterior chamber; the source of the bleeding is the iris root or ciliary body. Although hyphema can be caused by many medical conditions, the most common one is trauma. Among the causes of

**Image 15.6** Hyphaema



Courtesy of Allen Foster. Retrieved from <https://flic.kr/p/CPv1Nf>.

trauma, athletic injuries rank the highest. In children without underlying diseases, non-accidental injuries should come to mind. Bleeding itself does not usually

**Image 15.7** Hyphaema from blunt trauma.



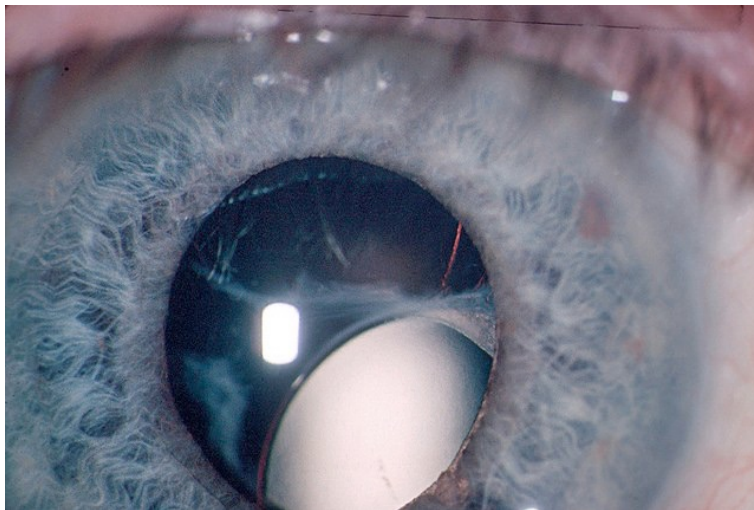
Some iris detail is visible. A level cannot be seen. Secondary glaucoma is likely until the blood has absorbed. Courtesy of International Centre for Eye Health. Retrieved from <https://flic.kr/p/dP3EPE>.

lead to loss of vision; however, the source of the bleeding is important. There is no correlation between the amount of bleeding and tissue damage; small amounts of bleeding can be observed in major injuries. When evaluating a patient with hyphema, the manner in which the trauma occurred, its intensity, and time should be determined. There is usually more than one eye pathology in patients with hyphema, and missing them could have serious consequences. For this



reason, these patients must be evaluated by an ophthalmologist. During emergency department treatment, bed rest, head elevation, eye patching, mydriatic/cycloplegic agents, antifibrinolytics (tranexamic acids), and anti-glaucoma drugs (beta blockers, carbonic anhydrase

**Image 15.8** Dislocated lens after trauma.



Courtesy of Bruce Noble. Retrieved from <https://flic.kr/p/9oxpTD>

inhibitors) can be used. Complications such as glaucoma, corneal blood staining, rebleeding, and optic atrophy can develop.

## Lens dislocation

Rupture of the fibrils attaching the lens to the ciliary body, following trauma can result in subluxation or dislocation of the lens. The most frequently posterior dislocations; due to the restrictive effect of the iris, anterior dislocations are less frequent. Sudden onset of loss of vision, monocular diplopia, photosensitivity, red eye, subconjunctival hemorrhage, and periorbital ecchymosis can be observed in these patients. Anterior dislocations can cause acute angle closure glaucoma by disrupting the flow of aqueous humor. USG and CT can be used for diagnosis. Treatment can vary from observation to surgical treatment, based on whether there are additional injuries and on the region of dislocation.

## Retinal Detachment

Traumatic retinal detachment (TRD) is the separation of the neurosensorial retina from the retinal pigment epithelia underneath it, leading to a disruption of its nourishment, and thus, a loss of vision. TRD typically develops months or

years after trauma. Although it can also be observed immediately after trauma due to distracting injuries and poor patient cooperation and poor visualization as a result of periorbital edema, it is difficult to diagnose at the acute stage. While the most common cause is closed blunt trauma, it has a higher incidence of open globe injuries. Symptoms include unilateral floaters, photopsia, and visual impairment. During the physical examination, visual acuity, visual field, and light reflex must be evaluated, and IOP should be measured. Retinal detachment and vitreous bleeding can be seen via ophthalmoscopy. Retinal detachment can be detected with USG, and a characteristic detachment pattern in the form of a “V” can be observed with CT. Early diagnosis is to ensure surgical intervention by an ophthalmologist. The prognosis depends on the size of the TRD, whether it involves the macula and length of time.

Vitreous hemorrhage: is bleeding in the posterior segment as a result of damage

to the retinal veins following blunt trauma. It is usually accompanied by retinal detachment, and the patient complains of a sudden loss of vision. Fundoscopy, USG, and CT can be used for diagnosis. Treatment is surgical; although there are differing views on its timing, due to high TRD frequency, it is suggested that the surgery is performed at an early stage.

## Periorbital Injuries

### Orbital Fractures

It can occur as isolated incidents, as well as together with other facial bone fractures. The most commonly observed isolated orbital fracture is a blow-out fracture. There are three theories regarding its formation: indirect impact related to increasing intraorbital pressure caused by trauma (hydraulic mechanism); direct conveying of energy during orbital wall trauma (the buckling mechanism); and a combination of the two mechanisms. The most common fractures are in the inferior and medial walls.

Examination findings can vary from a simple ecchymosis and edema to loss of vision. Sensitivity in the orbital wall, subcutaneous emphysema, and irregularity in orbital rhythms can be observed. Pupil diameters and light reflexivity must be evaluated. Upper and lower eyesight restriction and diplopia can develop if the inferior rectus and inferior oblique muscles are caught in the fracture line. In cases of medial wall fractures, patients can suffer epistaxis. When orbital pressure increases to very high levels, optic nerve damage and loss of vision can occur.

The gold standard in diagnosis is CT, and it should be used to evaluate the axial and coronal planes. Fractures can manifest in two ways in CT: the first one is direct visualization of irregularity and dislocations in bone cortexes, and the second one is visualization of air-liquid level in the sinuses around the orbit and air in the orbital cavity. Immediate surgery is rarely necessary for treatment. Surgical treatment indications include

enophthalmos greater than 2 mm, significant hypoglobus or diplopia, and an increase in orbital volume greater than 1 cm<sup>3</sup>.

### Orbital Compartment Syndrome

This condition is an ophthalmologic surgical emergency that develops following an acute increase in intraorbital volume and pressure. A sudden increase in pressure can cause blindness via compression of the optic nerves and/or vascular structures when not diagnosed at an early stage and treated. The most common causes are trauma, intraocular injections, and surgery. Orbital cellulite or abscess, orbital emphysema, foreign bodies, and tumors can also lead to this condition. Orbital volume is about 30 ml, and it is surrounded by the bony orbit, which prevents expansion. The only possibility is to expand toward the anterior, but that movement is limited by the canthal ligaments attached to the eyelids. Diagnosis is clinical. In patients with the predisposing causes mentioned above, it should be considered as a



possibility if there are findings such as reduced eyesight, diplopia, pain, and proptosis. Reduced eyesight, afferent pupil defect, elevated IOP, painful eye movement, and proptosis can be detected during a physical examination. Widened blind spot, reduced color sight (especially red), and afferent pupil defect detected during a visual field test are the most reliable findings that suggest optic nerve damage. The possibility of optic disc edema and retinal vein occlusion or congestion should be investigated with fundoscopy. If the patient history and physical examination support the findings, no time should be lost with visualization methods. Normal IOP level is 3–6 mmHg. Although there is not a specific pressure limit defined for orbital compartment syndrome, values  $\geq 30$  mmHg are considered to be high. The most important factor in making a treatment decision is the presence of clinical findings. Treatment is surgical, and lateral canthotomy and cantholysis are the surgeries preferred most often.

## Eyelid Lacerations

Eyelids anatomically consist of five layers. The outermost layer is the thin skin layer; beneath it is the subcutaneous tissue, and beneath that are the orbicularis oculi muscle, which allows the eyelids to be shut, the meibomian glands and the tarsal plate containing the eyelashes, and the innermost layer is the conjunctiva. Eyelid lacerations (ELs) are injuries caused by blunt or penetrating mechanisms. Because the eyelid is anatomically thin, it provides little protection against penetrating injuries, and the risk of globe injury is high in penetrating traumas. ELs are injuries that require special attention, and certain points must be considered. Before repairing the laceration, a complete physical examination must be undertaken. During the examination, lid and globe movements, visual field, corneal injuries, foreign bodies, and globe perforation should be evaluated. All patients must be asked about tetanus immunization. If there has been no immunization, immunoglobulin (250 U)

and toxoid should be administered together. Superficial lacerations can be sutured with 6.0 nylon or polypropylene. Eyelids have a risk of edema, and a cold compress after repair can decrease swelling and wound tension. Sutures can be removed on the fifth day. Ptosis in the eye, lacerations closer than 1 cm to the medial canthus, and cuts reaching the tarsal plate should be evaluated by an ophthalmologist or plastic surgeon.

## Retrobulbar hematoma

Retrobulbar hematomas are hemorrhages formed behind the globe due to trauma, surgery, and posterior injections. They are usually arterial in origin; the inferior orbital arteries and anterior ethmoidal arteries are most commonly injured. The clinical importance is that this condition leads to compartment syndrome by causing increased pressure inside the orbital cavity. Patients can suffer decreased visual acuity, sluggish light reflex, restricted eye movement, painful proptosis, and afferent pupil defect. CT is the most commonly preferred

visualization method for diagnosis. Although there is not a globally accepted algorithm for treatment, there are medical and surgical treatment options. Corticosteroids are used in medical treatments, and lateral canthotomy and cantholysis are the surgical treatments.

## Chemical Injuries

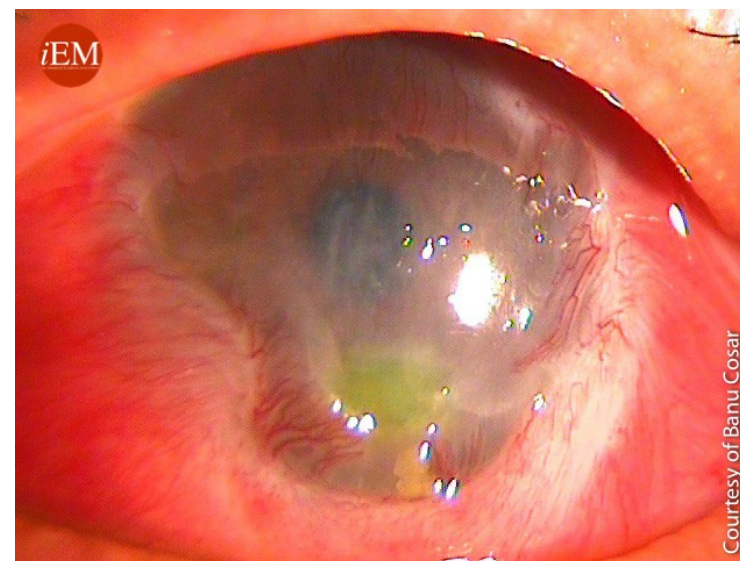
Eye traumas caused by chemical substances constitute a wide spectrum ranging from corneal abrasions, which are simple burn symptoms, to serious burns that can result in permanent blindness. The most commonly encountered chemicals are cleaning materials, personal care items, and automobile chemicals. Alkaline chemical injuries are more common than acidic ones. Because acidic materials lead to coagulation necrosis and scar formation, deep penetration is restricted. Alkaline materials cause deeper wounds due to liquefaction necrosis. Burns are grouped into four grades, based upon intensity.

Grade 1: Only epithelial damage; no limbal ischemia.

Grade 2: Obscurity on the cornea; however, iris details can be spotted and there is ischemia in less than 1/3 of the limbus.

Grade 3: Total loss of corneal epithelia. Stromal obscurity prevents spotting iris

**Image 15.9** corneal chemical burn



details. 1/3–1/2 limbal ischemia.

Grade 4: The cornea is completely opaque and there is >50% limbal ischemia.

While the prognosis is good for grades 1 and 2, it is poor for grades 3 and 4. The first thing to do when a chemical substance contacts the eye is to irrigate it with normal saline or Ringer's lactate solution in order to neutralize the eye's pH. Applying a local anesthetic will relieve the patient's pain. If care is being administered at the scene, tap water can be used for irrigation. Grade 1 and 2 injuries can be treated with antibiotics, steroids, and cycloplegic drugs. As antibiotics, preparations containing tobramycin or quinolone (ciprofloxacin, ofloxacin) can be used 4–5 times per day. Steroids decrease inflammation and prevent neutrophil activation. Grade 3 and 4 injuries may require surgical treatment.

**References and Further Reading,** click [here](#)

# The Red Eye

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by David Brian Wood

## Case Presentation

*A 27-year-old female with no past medical problems presents to the emergency room complaining of 2 days of a red, itchy and burning left eye. She notes that she has had a lot of watery discharge during this time and that her vision is blurry on occasion but improves after blinking a few times. She works in a day care where many of the children have been sick lately. She does not use corrective lenses and does not recall any trauma to the eye. She also denies systemic symptoms such as fever, photophobia, or joint pain. Vitals: T 98.5oF, HR 78, BP 124/68, RR14, SpO2 100% on room air. Visual acuity: 20/20 in both eyes. Peripheral fields: intact. Forehead/maxilla: no erythema or swelling. Lids/lashes: left eyelid mildly swollen. Otherwise, lids and lashes are normal, and eversion of eyelid demonstrates no foreign bodies. Conjunctiva: conjunctiva on the left is diffusely injected, and there is watery discharge.*

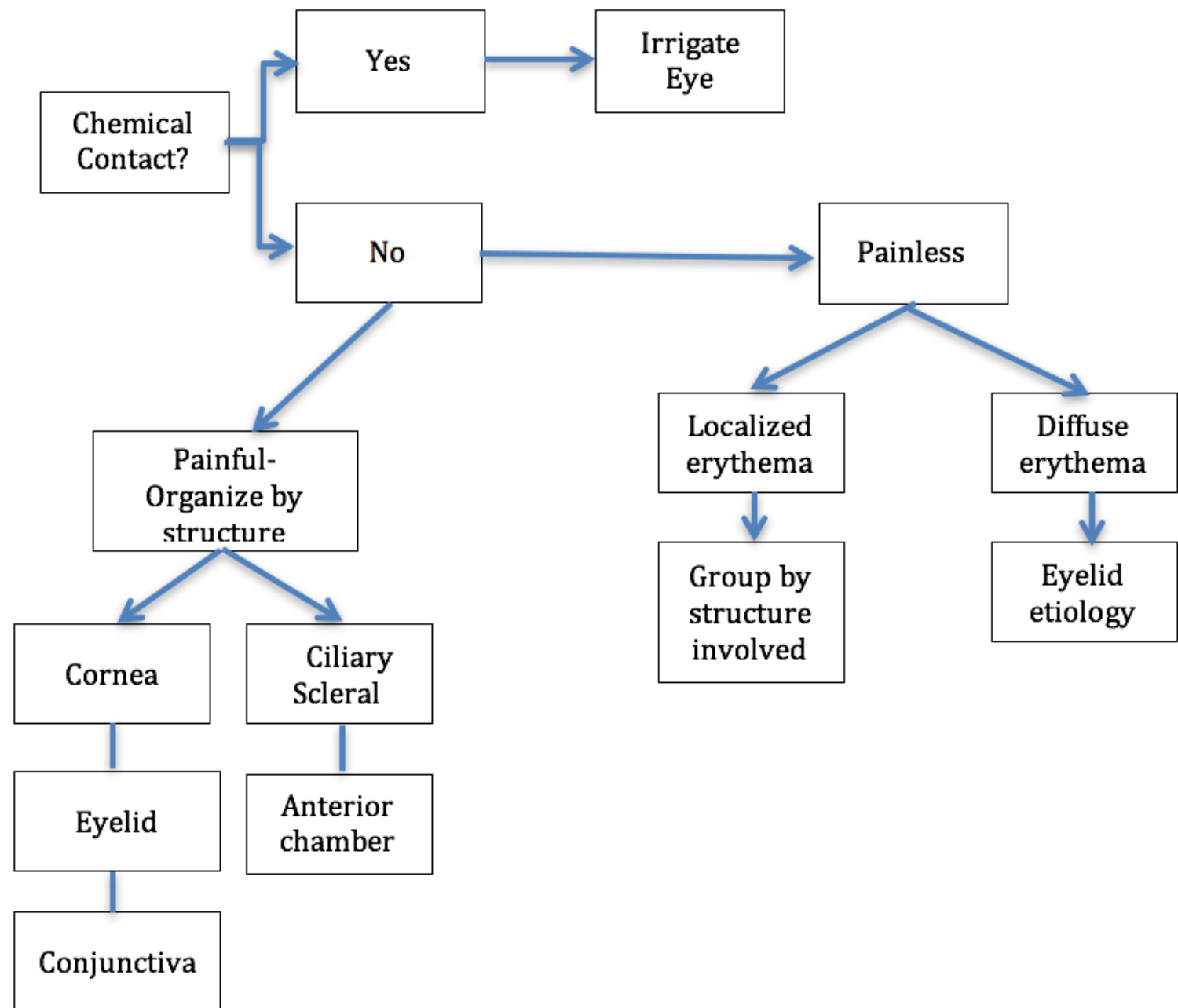
*Pupils: round, reactive to light, and equal bilaterally. Slit lamp: lids, lashes, and conjunctiva as above. No abrasions or lacerations visualized with fluorescein. Anterior chamber without cell and flare. Intraocular pressure: 18 mmHg bilaterally.*

## Critical Bedside Actions and General Approach

The initial step in the assessment is to determine if there is chemical exposure. The eye should be irrigated for at least 30 minutes or longer. If there has been exposure to an alkaline substance, irrigation can be stopped once the pH of the tears is neutral.

If there has been no chemical exposure, a thorough history and physical should be obtained. One practical approach is to first subdivide the etiologies into painful or painless.

**Figure 15.1** Approach to red eye.



**Flowchart 1:** Approach to the presentation of a red eye in the Emergency Department. Adapted from Life in the Fast Lane: The Red Eye Challenge.

*Approach to the presentation of a red eye in the Emergency Department. Adapted from Life in the Fast Lane: The Red Eye Challenge.*



## Differential Diagnoses

### Painful red eye

If the eye is painful, break it down based on the anatomic structure involved.

- Cornea (abrasions, ulcer, keratitis, herpes simplex keratitis)
- Eyelid (internal hordeolum, chalazion, external hordeolum, blepharitis, HZ ophthalmicus, preseptal cellulitis)
- Conjunctiva (conjunctivitis, dry eyes, glaucoma)
- Ciliary or scleral (scleritis, episcleritis)
- Anterior chamber (anterior uveitis/iritis, endophthalmitis, hyphema)
- Posterior chamber—usually does not cause a red eye and is more likely to interfere with vision or cause deep dull pain.

### Painless red eye

If the red eye is painless, determine if the erythema is localized or diffuse.

- Diffuse- usually caused by an eyelid issue such as blepharitis, ectropion, trichiasis, entropion, sty, or tumor.
- Localized- based on the structure involved. Examples include subconjunctival hemorrhage, trauma, corneal foreign body, chalazion, and pterygium.

## History and Physical Examination Hints

A thorough history should be obtained by paying particular attention to the following:

- Trauma to the face or eye (including possible foreign bodies)
- Pain and the specific location of the pain
  - Photophobia
  - Pain with extraocular movements
- Change in vision

- Systemic symptoms including fever, URI symptoms, weight loss, or joint pain

- History of inflammatory disease (SLE, MS, ankylosing spondylitis, etc.)

The patient's **description of eye sensation** can also help with the evaluation of the red-eye.

- Itching/burning: conjunctivitis, dry eye syndrome, or blepharitis.
- Foreign body sensation: corneal irritation or inflammation
- Sharp pain: anterior chamber process such as keratitis, uveitis, acute angle-closure glaucoma
- Dull pain: increased intraocular pressure or an extra orbital process

### Concerning findings on history:

- Severe ocular pain
- Persistently blurred vision

- Soft contact use-more susceptible to bacterial infection
- Immunocompromise

The ocular examination has numerous components, requires particular technical skill and therefore should be approached systematically so that no part of the exam is overlooked.

One approach to examining the eye is for the examiner to begin peripherally and progress inward, ending with the fundoscopic examination as shown below: **Order of Exam**

1. Visual Acuity : Remember to test each eye with and without corrective lenses. If they do not have corrective lenses, a pinhole can be used which will correct most refractive disturbances. If vision is too poor to read an eye chart, assess if the patient can detect the following: count fingers, detect hand motion, any light perception.

## 2. Peripheral fields

3. Extra-ocular eye movements: Look for disconjugate gaze and ask if the patient develops any diplopia when looking in a certain direction. Either of these findings suggests an entrapped extraocular muscle or nerve deficit.

4. Surrounding structures (forehead and maxilla): Assess for surrounding erythema, induration, or rash.

5. Eyelids and lashes: Remember to evert the eyelids as well. Look for localized swelling or redness.

## 6. Conjunctiva

7. Pupils: Note the size, shape, symmetry, clarity, and reactivity to light. Assess for an afferent papillary defect (APD) using the swinging flashlight test. If the pupil dilates when the light is shining in it, that eye has and APD and is not transmitting as strong of a response to the light compared to the opposite eye. An APD can be caused by many problems

including vitreous hemorrhage, retinal detachment, or optic neuritis.

8. Slit lamp exam: Re-examine the lids, lashes, conjunctiva, and cornea. Use fluorescein to evaluate for foreign bodies, abrasions, lacerations, or Siedels sign (streaming of aqueous humor from punctured globe site). Always have the patient move the eyes in all directions to visualize the entire conjunctiva. Visualize the anterior chamber to look for cell and flare, white blood cells (hypopyon), or red blood cells (hyphema).

9. Intraocular pressure assessment: Do not measure if there may be a penetrating injury as this would exacerbate aqueous humor leakage.

- A pressure of 10-20mmHg is considered normal.
- A pressure >20mmHg warrants an ophthalmology consult.
- Pressures >30 necessitate rapid treatment of underlying etiology (lateral

canthotomy for retrobulbar hematoma or medication administration for acute closed-angle glaucoma).

10. Fundoscopic exam: Often difficult to do a thorough exam in the emergency department, as the eye is typically not dilated.

*“The application of topical anesthetics to the eye helps to differentiate between superficial and deeper causes of pain. Pain that is not completely relieved by topical anesthetics is more likely to be from the sclera or anterior chamber.”*

## Emergency Diagnostic Tests and Interpretation

### Laboratory tests

In most cases, laboratory tests are rarely indicated or of much utility. If there is a

concern for an underlying autoimmune disorder or temporal arteritis erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) levels can be assessed.

### Imaging

The two most common scenarios of obtaining imaging of the red eye include facial fracture and penetrating foreign body. Plain radiographs can be obtained to assess for facial fractures. However, CT of the facial bones has replaced this imaging modality as it is more sensitive and specific for fractures. If there is a concern for a penetrating foreign body, a CT of the orbits can also be obtained. An MRI is an excellent modality for assessing foreign bodies but is contraindicated if there is any concern that the object may be metallic.

## Emergency Treatment Options

As stated previously, any eye that has come into contact with a chemical should be irrigated with 1-2L of NS until the pH of tears has returned to neutral. If there is

a concern for retrobulbar hematoma and the intraocular pressure is >40mmHg, a lateral canthotomy should be performed. If neither of these conditions exists then treatment can be tailored to the specific diagnosis.

## Painful Red Eye

### Cornea

#### Corneal abrasions

Most corneal abrasions will heal rapidly independent of intervention. Treatment focuses on preventing secondary infection and controlling pain. A significant component of the pain from a corneal abrasion comes from ciliary spasm that can be relieved by topical cycloplegics.

- Cyclopentolate is a short acting agent, Repeat every 4-6 hours.
- Homatropine is a long acting. Its effect lasts 2 days.

In addition, oral NSAIDs or opiates may be needed to control the pain adequately.

To prevent secondary infection, topical antibiotic ointments can be used (below recommendations are adopted from Harwood-Nuss and Rosen). If the abrasion is large or crosses the central visual axis, the patient should follow up with ophthalmology within 24 hours. Otherwise, follow-up can occur within 72 hours to ensure the abrasion is healing.

**Image 15.10** A corneal abrasion after staining with florescine.



Courtesy of James Heilman, MD – Own work, CC BY-SA 3.0, <https://commons.wikimedia.org/w/index.php?curid=11918476>

## Antibiotics

- Gentamicin ointment/solution 0.3%
- Ciprofloxacin solution 0.3% – Effective against *Pseudomonas*; prescribe to contact lens wearers
- Erythromycin ointment 0.5% – Effective against *Pseudomonas*; prescribe to contact lens wearers
- Ofloxacin 0.3%
- Polymyxin/trimethoprim

## Antivirals

- Trifluridine 1%
- Vidarabine 3%

## Corneal ulcers

Corneal ulcers are more serious and can pose a significant threat to the patient's vision. Ophthalmology should be consulted emergently for cultures of the ulcer and initiation of antibiotics as well as, in certain cases, antifungals.

**Image 15.11** Corneal ulcer with circumcorneal congestion.



Courtesy of P Vijayalakshmi. Retrieved from <https://www.flickr.com/photos/communityeyehealth/5444946687>

## Herpes Simplex Keratitis

Herpes Simples infections can be diagnosed based on its characteristic dendritic pattern seen with fluorescein staining. Conjunctival infections can be treated with trifluridine one drop up to nine times per day and antibiotic ointment such as erythromycin can be added to prevent secondary infections. All patients for which there is a concern for herpes keratitis should be seen by an ophthalmologist within 48 hours.



## Image 15.12 Herpes simplex virus



Top left: Child with measles and severe herpes simplex keratitis affecting the right eye. Top right: Dendritic ulcer stained with fluorescein dye Bottom left: Geographic ulcer stained with fluorescein dye Bottom right: Inflamed conjunctiva and geographic ulcer Photo (clockwise from top-left): John Sandford-Smith, Allen Foster, David Yorston). Retrieved from <https://www.flickr.com/photos/communityeyehealth/5616320250/in/photolist-9yi7kL-cAjBe7-dQmoJB-9yi7rQ-cAjAUY-cAjAQj-cAjBbA-dQmoR2-cAjB8s-9EwFS5-9AaetU-9wqCZN-cAjAAC>

## Eyelid

### Internal/External hordeolum and acute chalazion

Initial treatment for these conditions consists of warm compresses and erythromycin ointment twice daily for 7-10 days. Referral to ophthalmology as an outpatient can be made if symptoms

do not improve after 1-2 weeks of treatment.

## Image 15.13 Chalazion



© International Centre for Eye Health [www.iceh.org.uk](http://www.iceh.org.uk), London School of Hygiene & Tropical Medicine. Retrieved from <https://www.flickr.com/photos/communityeyehealth/8423448539/in/photolist-dQmNx6-dQmnMn-9Ezoru>

## Acute Blepharitis

Blepharitis is caused by inflammation of an eyelash follicle due to an overgrowth of bacterial skin flora. The mainstay of treatment consists of daily cleaning of the edge of the eyelashes.

## Image 15.14 Posterior blepharitis



Courtesy of John KG Dart Retrieved from <https://www.flickr.com/photos/communityeyehealth/32271350840/in/photolist-aBgGJh-aBgGZo-aBe1Hi-aBgGR1-aBe1cn-aBgFWu-aBgH3S-aBe1gM-aBgFV9-aBe1We-9J2LcJ-9J2L9d-9A9VAo-9HYUGv-RaH7r9-RaH7hG>

## Herpes zoster ophthalmicus

Patients with herpes zoster ocular infections should be treated with artificial tears and erythromycin ointment to prevent secondary infection. Oral antiviral medication can be used if there is skin involvement and, after consultation with an ophthalmologist, topical antivirals may be prescribed as well. The significant pain from herpes zoster infections may require opiate treatments or the use of an

antidepressant such as amitriptyline 25mg P.O. TID.

**Image 15.15** Herpes zoster ophthalmicus



Courtesy of John Sandford-Smith. Retrieved from <https://www.flickr.com/photos/communityeyehealth/5686390813/in/photolist-9yKTSq-9EueSV-9yxBrw>

## Conjunctiva

### Acute closed-angle glaucoma

The overall goal is to reduce intraocular pressure by decreasing aqueous production and increasing outflow. Aqueous outflow is improved through miosis as this pulls the iris away from the trabecular meshwork. Definitive treatment is an iridectomy performed by an ophthalmologist.

**Image 15.16** Acute glaucoma, red eye.

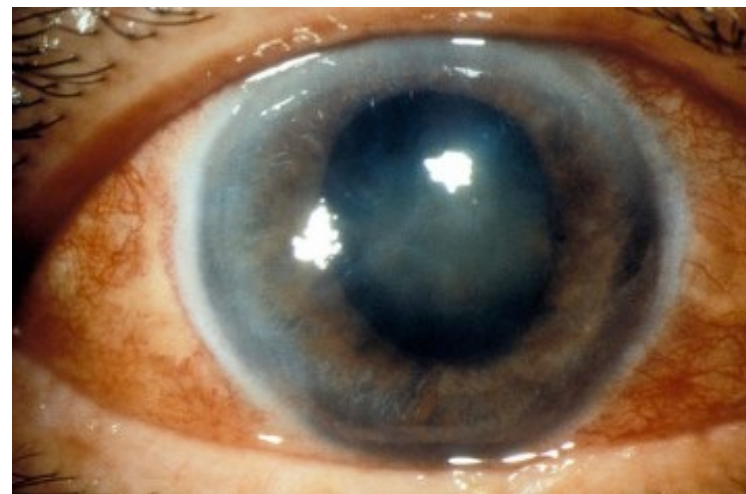


Photo: International Centre for Eye Health [www.iceh.org.uk](http://www.iceh.org.uk), London School of Hygiene & Tropical Medicine.

Acute closed-angle glaucoma and IOP lowering agents can be divided into 2 category (adopted from Tintinalli)

### Topical agents

- Timolol – 0.5% 1 gtt q5min x3 doses then 1gtt q12h, decreases production of aqueous humor. Should be avoided in patients with asthma, heart block, and heart failure.
- Pilocarpine – 2% 2 gtt q5min until pupil constricts. Then 1gtt q6h. It is outflow

parasympathetic agonist, causes myosis. Rarely causes sweating, bradycardia, hypotension.

- Apraclonidine – 1% – 1gtt q5min x3 doses. Decreases production of aqueous humor (alfa-2 agonist). Used most often in chronic glaucoma but may be useful in AACG.
- Latanoprost – 0.005% 1gtt daily. Increases outflow.

### Systemic agents

- Acetazolamide – 500mg IV q12h or 500mg PO q6h. It is a carbonic anhydrase inhibitor. Decreases production of aqueous humor. It should be avoided in patients with respiratory disease as it causes respiratory acidosis.
- Mannitol 20% – 1-2 gram/kg IV over 30-60 minutes. Decreases Aqueous humor by increasing serum osmolality.



## Conjunctivitis

Most cases of conjunctivitis will be due to allergic or viral causes and can be treated with artificial tears 5-6 times per day. If there is a concern for a bacterial cause of conjunctivitis the patient can be treated four times daily for 5-7 days with topical antibiotic drops such as trimethoprim or polymyxin B. If the patient wears soft contact lenses, then Pseudomonal coverage is necessary with a fluoroquinolone or aminoglycoside.

**Image 15.17** Lorem Ipsum dolor amet, consectetur



Courtesy of Rbmorley – Robert Morley,  
Retrieved from <https://commons.wikimedia.org/w/index.php?curid=8658905>

## Ciliary/scleral

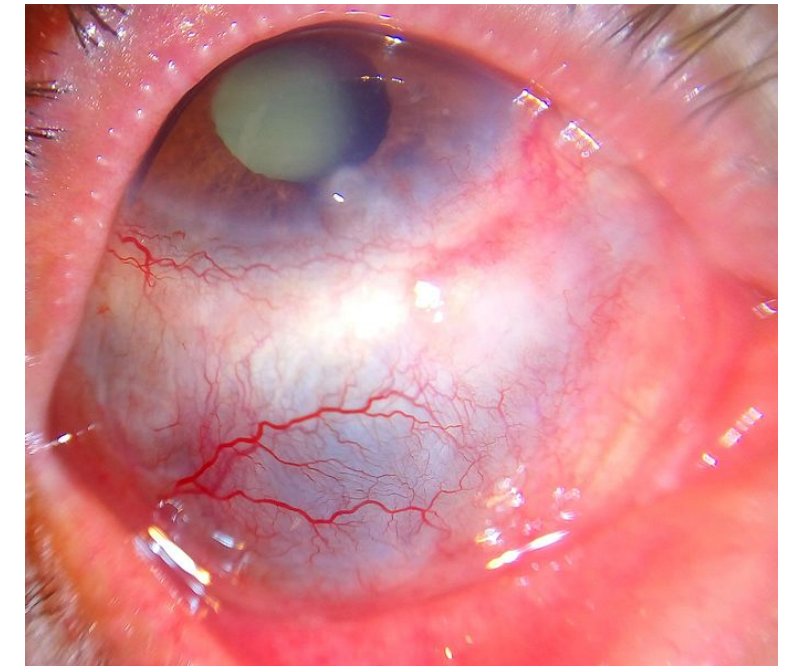
### Episcleritis

Artificial tears can be used up to four times per day to help lubricate the eye. A trial of oral NSAIDs can be given in the emergency room and if pain resolves can be continued as an outpatient. If the patient continues to have significant pain after NSAID a topical steroid can be used to relieve the discomfort. The steroid drops can be continued as an outpatient until seen by ophthalmology in 2-3 weeks.

### Scleritis

Oral NSAIDs can be used for pain control as in episcleritis. Topical steroids are ineffective in scleritis, however, and oral steroids may be used starting at prednisone 60mg daily for 1 week and a slow taper over the next 4-6 weeks. Ophthalmology should be consulted and may recommend starting additional immunosuppressive agents.

**Image 15.18** Recurrent scleritis



By Imrankabirhossain – Own work, CC BY-SA 4.0, Retrieved from <https://commons.wikimedia.org/w/index.php?curid=60068874>

## Anterior chamber

### Uveitis/Iritis

Often related to a systemic process such as a rheumatologic condition, malignancy, or infection. Iritis and Uveitis can be treated symptomatically with cycloplegics, which paralyze the ciliary body and pupillary sphincter. A long-acting agent such as homatropine will last for 2-3 days after one dose and can control the pain until the patient can be

seen by an ophthalmologist. These patients should be seen by an ophthalmologist within 48 hours.

**Image 15.19** Acute anterior uveitis



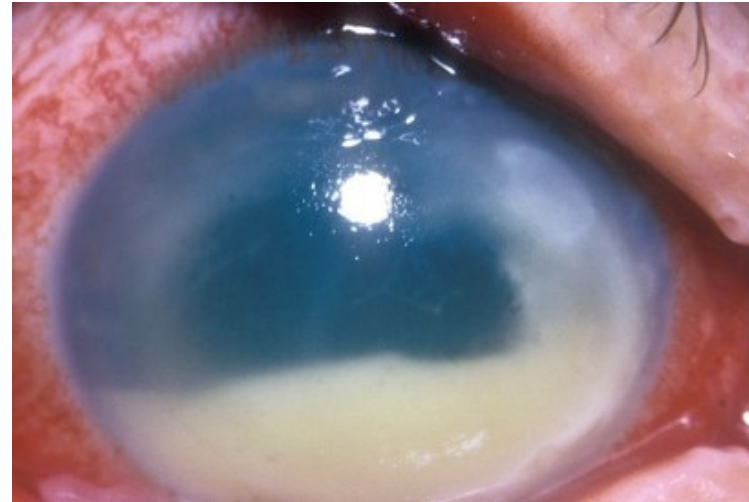
45-year-old female. Complains of painful eye and discomfort in bright light with watery discharge. VA 6/12. Photo: International Centre for Eye Health [www.iceh.org.uk](http://www.iceh.org.uk), London School of Hygiene & Tropical Medicine. Retrieved from <https://www.flickr.com/photos/communityeyehealth/5594571189/in/photolist-9wnD6X-czQdY9>

## Endophthalmitis

Usually leads to vision loss and therefore requires an emergent ophthalmology consult. Admission is necessary to administer IV antibiotics. In addition, the ophthalmologist may aspirate the vitreous

and administer intraocular antibiotics and steroids.

**Image 15.20** Endophthalmitis



Endophthalmitis with extensive hypopyon consistent with active infection. © International Centre for Eye Health [iceh.lshtm.ac.uk](http://iceh.lshtm.ac.uk), London School of Hygiene & Tropical Medicine. Retrieved from <https://www.flickr.com/photos/communityeyehealth/7608314920/in/photolist-9oAtpo-cAjAXs-cAjAHs-9Ex9Yq-MGPFgy>

## Hyphema

Initial treatment consists of elevating the patient's head to allow the red blood cells to settle inferiorly where they are less likely to obscure the trabecular meshwork and raise intraocular pressure. If intraocular pressure is increased >30mmHg, the same treatment options can be employed as described in

glaucoma. Patients with hyphema should have ophthalmology consult in the

**Image 15.21** Hyphema



By Rakesh Ahuja, MD – Own work, CC BY-SA 2.5, <https://commons.wikimedia.org/w/index.php?curid=1270407>

ED.

## Others

### Retrobulbar hematoma

In patients in which there is a concern for retrobulbar hematoma or other space-occupying retro-orbital lesions in which the intraocular pressure is >40mmHg, a lateral canthotomy should be performed to relieve the pressure and spare the



optic nerve from further damage. If a retrobulbar hematoma is present, but the intraocular pressure is below 40mmHg, and there is no vision loss, ophthalmology should be consulted. Similar medications to those used in acute closed-angle glaucoma can also be used to help decrease the intraocular pressure.

**Image 15.22** Subconjunctival Hemorrhage



By Daniel Flather – Own work, CC BY-SA 3.0, Retrieved from <https://commons.wikimedia.org/w/index.php?curid=15651313>

## Painless Red Eye

## Subconjunctival Hemorrhage

No treatment is necessary for a subconjunctival hemorrhage as the blood

**Image 15.23** Pterygium



By unknown photographer. Retrieved from <https://commons.wikimedia.org/w/index.php?curid=56208402>

will resolve within 2 weeks.

## Pterygium

The management of a pterygium focuses largely on preventing it from enlarging through the use of sunglasses or goggles to prevent ultraviolet light, dust, and other irritants. If the pterygium is inflamed, artificial tears can be used four times per day. For severe inflammation, topical steroids can be used (below

recommendations adopted from Harwood-Nuss) or topical NSAIDs. Surgical removal by an ophthalmologist may be indicated if the visual axis is impaired or for persistent irritation.

## Topical steroids

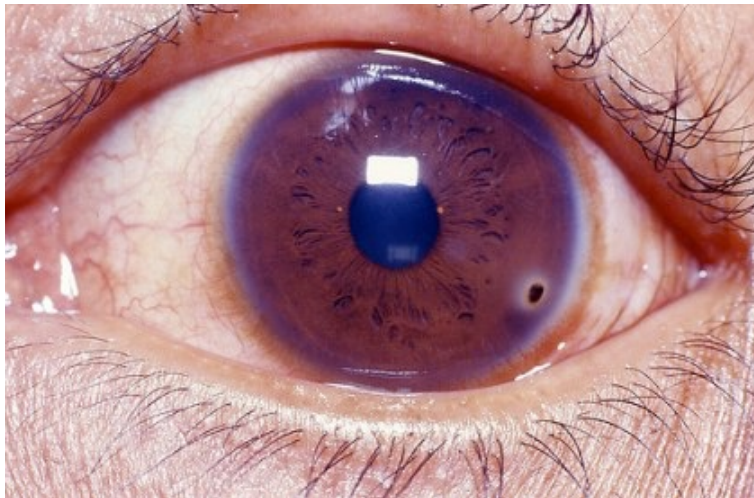
- Prednisolone acetate 0.125% – Potent. Effective for anterior chamber inflammation
- Medrysone 1% – Mild potency. Use for allergies
- Rimexolone 1% – Mild potency
- Dexamethasone 0.1% (solution), 0.05% (ointment) – Potent

## Corneal foreign body

If a small corneal body is seen on the exam with the slit lamp, the eye should first be anesthetized prior to removal with a topical anesthetic. The foreign body can then be removed using a small-gauge needle, fine forceps, or irrigation. A metallic foreign body will usually leave a rust ring that should be removed with an

ophthalmic burr if available. After the foreign body is removed, the resulting

**Image 15.24** Corneal foreign body



*This is a 'rust ring' which shows signs of having been present for some days. The iron particle or 'rust' will lift off the cornea easily but will leave a stained area beneath. Removal with a needle or drill (burr) will be necessary. Retrieved from <https://www.flickr.com/photos/communityeyehealth/8408519738>*

defect can be treated as a corneal abrasion with topical antibiotic ointment.

## Procedures

While rare, the main ophthalmologic procedure performed in the emergency department is a lateral canthotomy. The overall goal of the lateral canthotomy is too severe the connection between the

bony orbit and lower eyelid to allow the orbit to move forward to compensate for the increased pressure placed on it. To perform a lateral canthotomy the canthus is first anesthetized, then crushed with curved forceps, and then cut with scissors. The inferior canthus tendon can then be identified by strumming it with the scissors and can then be incised. Following severing the tendon, the inferior eyelid should be released completely from the orbit. Depending on the amount of time the retina was ischemic, there may be rapid improvement in vision as the pressure is reduced.

## Pediatric Patients

Pediatric patients suffer from many of the same etiologies of red eyes as adults but may be more difficult to obtain an adequate exam on. Visual acuity in children begins around 20/100 and improves to 20/20 by approximately 8 years of age. Before 5 years of age, most children will be unable to read a Snellen chart. Visual acuity can be grossly tested by covering each eye separately and

having the child fixate on an object of interest. If the vision is normal, the child will continue to fixate on the object. By around 3 years old visual acuity can be tested more effectively using an Allen chart or Tumbling E chart.

Neonatal conjunctivitis is usually caused by exposure to an infectious agent while exiting the birth canal. Most commonly neonatal conjunctivitis is caused by Chlamydia trachomatis or Neisseria gonorrhoea but can also be due to herpes simplex virus. Treatment is based on the incubation period as shown below (adopted from Harwood-Nuss). In addition, neonates with HSV will require ophthalmology consultation. HSV and gonococcal conjunctivitis will require inpatient therapy with ophthalmology serving as a consult.

N. gonorrhoeae has 2-7 days incubation period. Ceftriaxone 25-50mg/kg IV or IM, once is effective for treatment. For prophylaxis, 1% silver nitrate, 0.5%

erythromycin ointment, 1% tetracycline can be used.

C. trachomatis has 5-14 days incubation period. Treatment is erythromycin 50mg/kg/day divided q.i.d. PO x14 days. There is no prophylactic agent recommendation.

HSV has 1-2 weeks incubation period. Acyclovir 60 mg/kg/day divided q.i.d. for 2-3 weeks AND atypical anti-viral agent are used for treatment. There is no prophylactic agent recommendation.

## Disposition Decisions

The vast majority of patients presenting for red-eye will be discharged home. Even many of the ocular emergencies will be able to be discharged following evaluation by ophthalmology. There are a few conditions requiring admission such as endophthalmitis, retrobulbar hematoma, and globe rupture. Disposition and urgency of consultation are covered in the above clinical management of ocular problems.

**References and Further Reading**, click [here](#)



## Chapter 16

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# Selected Procedures





# Automated External Defibrillator (AED) Use

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by Mehmet Ali Aslaner

## Introduction

AED is a portable electronic device that produced to detect and treat the life-threatening cardiac arrhythmias such as like ventricular fibrillation and ventricular tachycardia in case of sudden cardiac arrest (SCA). AED defibrillate (electrical therapy) the patient to stop life-threatening arrhythmias and allow an effective rhythm.

AED device gives simple orders and can be used by a layperson who was previously trained before, a certified first responder, and health care professionals.

## Indication

2015 European Resuscitation Council Guidelines recommend using an AED for adult basic life support (BLS). It

should be used in combination with cardiopulmonary resuscitation (CPR). Pulseless ventricular tachycardia and ventricular fibrillation are the most common and treatable causes of SCA. Therefore, the use of an AED is very important and vital.

## Where to find an AED?

Public areas, corporate or government, should keep an AED in offices, shopping centers, airports, airplanes, restaurants, casinos, hotels, sports stadiums, community centers, fitness centers, health clubs, theme parks, schools and universities, workplaces and any other location where people may gather.

## AED Kit

An AED kit contains

- a face shield to provide a barrier between the patient and first aid provider during rescue breathing,
- rubber gloves,
- trauma shears for cutting through a patient's clothing to expose the chest,
- a towel for wiping away any moisture on the chest, and
- a razor for shaving extensively hairy chests.

## How to use an AED?

1. recognize abnormal status in case of BLS. When facing an unconscious person, you should decide if he/she is alive or not (unresponsive and not breathing normally) by the BLS algorithm.
2. If the person is not breathing normally, call the emergency services and send someone to get AED.
3. begin chest compressions with rescue breaths 30-2 (if trained or able to do).

4. As soon as the AED arrives,

- switch on the AED and attach the electrode pads on the patient's bare chest.
- CPR should be continued while electrode pads are being attached to the chest, if there is two rescuer.
- Follow the spoken/visual directions,
- ensure that no one is touching the patient while the AED is analyzing the rhythm.
- If a shock is indicated, push the shock button as directed (fully automatic AEDs will deliver the shock automatically). Immediately restart CPR 30-2 and continue as directed by the voice and visual directions.
- If no shock is indicated, continue CPR until emergency medical service (EMS) arrives.

Please watch the [video](#)

CPR providers should continue CPR with minimal interruption of chest compressions while attaching an AED. Standard AEDs are suitable for use in children older than 8 years. AEDs are safe to use. Currently, There are no published reports of AEDs' harmful effects on bystanders. Also, there are no reports of AEDs delivering inappropriate shocks. If someone has a sudden cardiac arrest, using an AED and giving CPR can improve the person's chance of survival.

**References and Further Reading,** click [here](#)

# Arterial Blood Gas (ABG) Sampling

by Matija Ambooz and Gregor Prosen

## Case Presentation

*A 23 years old pregnant woman was admitted with a history of polyuria, dysuria, fever, and thirst. She is an insulin dependent diabetic patient. She is febrile. Her chest is clear, and circulation is adequate. Urinalysis shows the presence of ketones, glucose, and leukocytes. Her lab results on admission are:*

*Na<sup>+</sup> 136 mmol/L, K<sup>+</sup> 4.8 mmol/L, Cl<sup>-</sup> 101 mmol/L, Glucose 23.2 mmol/L, Urea 8.1 mmol/L, Creatinine 0.09 mmol/L*

*Her ABG results are:*

*pH: 7.26*

*pCO<sub>2</sub> = 14 mmHg*

*pO<sub>2</sub> = 133 mmHg*

*HCO<sub>3</sub><sup>-</sup> = 7.1 mmol/L*

*Low  $p\text{CO}_2$  and low  $\text{HCO}_3^-$  indicates metabolic acidosis. Hyperglycemia, glycosuria, and ketonuria indicate DKA. There might be an underlying UTI that triggered DKA. Respiratory alkalosis is a compensation.*

## Introduction

Arterial blood gas (ABG) analysis is an important investigation to monitor the acid-base balance of critically ill patients.

ABG help to determine treatment may indicate the severity of the condition and can help to diagnose a disease. The respiratory status and acid-base equilibrium of individuals with pulmonary disorders, drug overdose, and metabolic disorders may be evaluated through this procedure.

Blood is drawn from a peripheral artery via single percutaneous needle puncture, or from an indwelling arterial cannula or catheter for multiple samples.

Partial pressures of carbon dioxide ( $\text{PaCO}_2$ ) and oxygen ( $\text{PaO}_2$ ), hydrogen ion activity (pH), total hemoglobin (Hb), oxyhemoglobin saturation ( $\text{HbO}_2$ ), and carboxyhemoglobin (COHb) and methemoglobin (MetHb) are directly measured.

Oxygen ( $\text{O}_2$ ) and carbon dioxide ( $\text{CO}_2$ ) are the most important respiratory gases, and their partial pressures in arterial blood show the overall adequacy of gas exchange. pH, which measures hydrogen ion activity, is a regular part of every arterial blood gas sampling (Image 16.1). To learn how to evaluate ABG analysis please click [here](#).

**Image 16.1** An example of an arterial blood gas analysis result.

ACID/BASE		37.0 °C
pH	7.329	
$p\text{CO}_2$	9.61↑	kPa
$p\text{O}_2$	8.24↓	kPa
$\text{HCO}_3^-$ act	37.1	mmol/L
$\text{HCO}_3^-$ std	31.6	mmol/L
$\text{Cl}^-$ (3)	8.1	mmol/L
BE (ecf)	11.1	mmol/L
ct $\text{CO}_2$	39.3	mmol/L
OXYGEN STATUS		37.0 °C
$\text{O}_2$ SAT(est)	89.1	%
ELECTROLYTES		
$\text{Na}^+$	145.9↑	mmol/L
$\text{K}^+$	4.71	mmol/L
$\text{Ca}^{++}$	1.26	mmol/L
$\text{Ca}^{++}$ (7.4)	1.22	mmol/L
$\text{Cl}^-$	94↓	mmol/L
AnGap	19.5	mmol/L
METABOLITES		
Lac	0.87	mmol/L
$p\text{Atm}$	94.3	kPa
↓, ↑ = Out of range		

Courtesy of Matija Ambooz



# ABG Sampling Procedure

## Indications

- to evaluate ventilation, (PacO<sub>2</sub>) acid-base status (pH and PaCO<sub>2</sub>), oxygenation status (PaO<sub>2</sub> and SaO<sub>2</sub>), and the oxygen-carrying capacity of blood (PaO<sub>2</sub>, HbO<sub>2</sub>, Hbtotat, and dyshemoglobins)
- to quantitate the patient's response to therapeutic intervention and/or diagnostic evaluation (e.g., oxygen therapy, exercise testing)
- to monitor the progression and severity of the observed disease. We usually evaluate these parameters in patients with multi-organ failure, both chronic and acute respiratory failure, ventilated patients, critically ill trauma patients, septic patients, patients with burns and poisoned patients.

## Contraindications

- Inadequate circulation,
- Burger's disease,

- Raynaud's syndrome,
- Full-thickness burns. Relative contraindications include:
- Skin infection at the site of puncture,
- Previous surgery in the area,
- Inadequate collateral flow,
- Partial-thickness burns,
- Atherosclerosis,
- Anticoagulation or coagulopathy\*.

\*ABG sampling can be performed safely in patients who are on anticoagulants or have other coagulopathies. In patients with severe disseminated coagulopathies, extreme caution is required.

## Equipment and Patient Preparation

Equipment used in arterial puncture include;

ABG syringe, for an adult, use a 20-gauge, 2.5-inch needle for a femoral sample and a 22 gauge, 1.25-inch needle

for a radial artery puncture, Also 23 gauge and 25 gauge needle can be used. A 23 gauge syringe may be used as it allows faster filling than 25 gauge one, but does not affect more pain to the patient (Figure 16.2).

**Image 16.2** Needle and syringe before assembly



- 70% isopropyl alcohol or an antiseptic solution,
- gauze or cotton-wool ball to be applied over puncture site,
- well-fitting non-sterile gloves
- puncture-resistant container.

With an adult patient who is conscious, follow the steps below (adapted from

W.H.O. best phlebotomy practice guidelines).

- Introduce yourself to the patient and ask their full name.
- Check that the laboratory form matches the patient's identity.
- Ask whether the patient has allergies, phobias or has ever fainted during previous injections or blood draws.
- Discuss the procedure and obtain verbal consent.
- If the patient is afraid or anxious, help him relax and make him more comfortable.
- Make the patient comfortable in a supine position.
- Place a clean paper or towel under the patient's arm.

## Procedure Steps

Various arteries can be used for blood collection. The radial, brachial, and femoral arteries are the sites most

commonly punctured for blood gas sampling in adults. The first choice is the radial artery due to its superficial anatomical location. It has good collateral circulation and is not surrounded by structures that could be easily damaged by puncturing.

The procedure as defined by W.H.O. guidelines consists of 16 steps for radial artery puncture.

1. Approach the patient, introduce yourself and ask the patient to state their full name.
2. Place the patient on their back, lying flat. Ask the nurse for assistance if the patient's position needs to be altered to make them comfortable. If the patient is clenching their fist, holding their breath or crying, this can change breathing and thus alter the test result.
3. Locate the radial artery by performing an Allen test for collateral circulation (Video 1). If the test fails to locate the radial artery or collateral flow is

inadequate, repeat the test on the other hand.

- **Video 1:** Modified Allen test; Radial and Ulnar Artery are both pressed to prevent blood flow. Ulnar artery is released after the hand becomes pale. If the hand flushes after 5s – 15s, the ulnar artery has sufficient blood flow and radial artery may be punctured. If it takes more than 15s for hand to flush, the ulnar artery has inadequate blood flow and this hand should not be punctured.
4. Perform hand hygiene, clear off a bedside work area and prepare supplies.
  5. Disinfect the sampling site on the patient with 70% alcohol and allow it to dry.
  6. Assemble the needle and heparinized syringe and pull the syringe plunger to

the required fill level recommended by the local laboratory. (1 – 3 mL)

**Image 16.3** Syringe and needle prepared for puncturing.



7. Holding the syringe like a dart, use the index finger to locate the pulse again, inform the patient that the skin is about to be pierced then insert the needle at a 45-degree angle, approximately 1 cm distal to the index finger, to avoid contaminating the area where the needle enters the skin.

8. Advance the needle into the radial artery until a blood flashback appears, then allow the syringe to fill to the appropriate level. DO NOT pull back the syringe plunger.

9. Withdraw the needle and syringe; place a clean, dry piece of gauze or cotton wool over the site and have the patient or an assistant apply firm pressure for sufficient time to stop the bleeding. Check whether bleeding has stopped after 2–3 minutes.

10. Activate the mechanisms of a safety needle to cover the needle before placing it in the ice cup.

11. Expel air bubbles, cap the syringe and roll the specimen between the hands to gently mix it. Cap the syringe to prevent contact between the arterial blood sample and the air, and to prevent leaking during transport to the laboratory.

12. Label the sample syringe.

13. Dispose appropriately of all used material and personal protective equipment.

14. Remove gloves and wash hands thoroughly with soap and water, then dry using single-use towels;

alternatively, use alcohol rub solution. Check the patient site for bleeding and thank the patient.

15. Check the patient site for bleeding and thank the patient.

16. Transport the sample immediately to the laboratory, following laboratory handling procedures.

ABG Sampling [video 1](#) and [video 2](#)

## Hints and Pitfalls

- Precooling proved to be useful for patients who had problems with anxiety and pain due to arterial blood puncturing. Cryoanalgesia can be provided by ice bag applied to wrist 3 minutes prior to arterial puncture.
- ABG measurements are particularly vulnerable to pre-analytic errors. Problems include air bubbles, improper anticoagulation, delayed analysis, non-arterial samples and other transport or handling related problems.

- After collection, the sample should be analyzed quickly. If a delay of more than 10 minutes is anticipated, the sample must be embedded in an ice bath. Leukocytes and platelets continue to consume oxygen in the sample after it is drawn and can cause a significant fall in PaO<sub>2</sub> over time at room temperature, especially in the setting of leukocytosis or thrombocytosis. Cooling decrease the metabolic activity of leukocytes and platelets and thus prevent the clinically important effect of oxygen consumption for at least 1 hour.
- Room air has a PO<sub>2</sub>, of approximately 150-160 mmHg (at sea level) and a PCO<sub>2</sub> of essentially zero. Thus, air bubbles that mix and equilibrate with arterial blood will shift the PaO<sub>2</sub> toward 150 mmHg and PaCO<sub>2</sub> toward zero.
- Heparin must be added to the syringe as an anticoagulant. Because the pH of heparin is near 7.0, and the PO<sub>2</sub> and PCO<sub>2</sub> of the heparin solution are near room air values, excess heparin can

alter all three ABG measurements. After flushing the syringe with heparin, a sufficient amount usually remains in the dead space of the syringe and needle for anticoagulation without distortion of the ABG determination.

## Post Procedure Care and Recommendations

After collection of at least 1 to 2 mL of sample, the needle is removed, and firm pressure is applied at the site of puncture for 3 – 5 minutes. If the patient is on anticoagulants or has any coagulopathy, the pressure is required for 10 – 15 minutes.

## Complications

There are some potential complications related to arterial blood sampling.

- Temporary arterial occlusion and spasm may be prevented by helping the patient relax. One can achieve this by explaining the procedure, positioning the patient comfortably and using precooling or other forms of analgesia.

•The hematoma is a common complication without a serious sequel. It may be prevented by inserting the needle without puncturing the fat side of the vessel and by applying firm pressure at the site of a puncture. Due to high pressure present in arteries, pressure should be applied for a longer time than in venipuncture.

- Nerve damage may be prevented by choosing an appropriate site for puncturing and avoiding redirection of the needle.
- Fainting may be prevented by ensuring that the patient is lying down with their feet elevated.

## Geriatrics, Pediatrics, Pregnant Patients and Other Considerations

Pediatrics should be mentioned as a special consideration due to the challenge that they present in the form of obtaining vascular access and blood samples. Fear and anticipation of pain associated with procedures may the



hospital experience traumatic for children. The procedure should be explained before starting and consent taken. Parents may provide comfort to the child, but there is also a potential for parents to faint. Products to decrease the pain may be considered in stable patients. Capillary blood obtained from heel is another option and can be used for gas analysis when arterial access is unavailable or when the clinician is not comfortable obtaining a percutaneous arterial blood sample. Arterial blood may be obtained from radial, brachial, dorsalis pedis and in newborn infants, the umbilical arteries. The radial artery is the site of choice. For arterial puncture in infants and children, a small-gauge butterfly needle is preferable to a needle and syringe as used in adults. In contrast to arterial puncturing in adults, continuous, but gentle suction should be provided in infants. Pulsating blood is a good sign that the radial artery has been punctured.

**References and Further Reading,** click [here](#)

# Arthrocentesis

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by Tanju Tasyurek

## Introduction

Arthrocentesis is an acknowledged, useful procedure to puncture and aspiration of a joint. It is usually performed both as a diagnostic and therapeutic tool for various clinical situations. Arthrocentesis (synovial fluid aspiration) of a joint can be performed either diagnostically (for identification of the etiology of acute arthritis) or therapeutically (for pain relief, drainage of effusion, or injection of medications). Arthrocentesis is required procedure in majority of patients with monoarthritis and is mandatory if an infection is suspected.

## Indications of Arthrocentesis

- Diagnosis of septic or crystal-induced arthritis

- Evaluation of therapeutic response for septic arthritis
- Diagnosis of traumatic bony or ligamentous injury
- Installation of medications for acute or chronic arthritis
- Relief of the pain of acute hemarthrosis
- Determination of communication between the laceration and joint space

## Contraindications

- Absolute contraindication to arthrocentesis is an infection in the tissue overlying the site to be punctured. However, inflammation with warmth, swelling, and tenderness may overlie an acutely

arthritic joint, and this condition may mimic a soft tissue infection.

- Coagulopathy is an absolute contraindication. However, few studies are demonstrating whether it is dangerous performing arthrocentesis in patients using anticoagulants. It was found safe even in those who have international normalized ratios as high as 4.5.
- Prosthetic joints increase the risk for infection. Therefore arthrocentesis should be avoided for these joints. However, if an infected prosthesis is suspected, arthrocentesis should be performed.

## Equipment

- Sterile gloves and drapes
- Gauze pads (5), 4 × 4 inches.
- Skin cleaning agent
- Local anesthetic such as Lidocaine 1%

- Various syringes (5 mL, 20 mL, 30 mL, 60 mL)
- Various size of needles, 18 or 20 G and 25 or 27 G
- Morbidly obese patients might require a 21-gauge spinal needle for arthrocentesis
- Specimen tubes
- Bandage

## General Arthrocentesis Technique

Arthrocentesis is a relatively simple procedure. Knowledge of anatomic landmarks and patient positioning will aid in the successful completion of joint aspiration. Defining the anatomy is the most important part of the procedure. The clinician should be familiar with the anatomy of the specific joint and landmarks in order to avoid puncture of tendons, blood vessels, and nerves.

The procedure should be explained to the patient and written consent should

be taken. To avoid infection, aseptic technique is essential, including the use of sterile gloves and instruments. After skin preparation with antiseptic solutions, the clinician should allow the solution to dry for several minutes because the bactericidal effects of iodine are dependent on both concentration and time. Iodine solution should be removed with an alcohol sponge. This will prevent iodine transfer into the joint space, which can cause an inflammation.

Without anesthesia, arthrocentesis may be quite painful. Entire route of the needle should be anesthetized from skin to joint capsule. 1% or 2 % lidocaine can be used.

Rigid needles are preferred whereas some clinicians can use sturdy catheters. As a general rule, one should try to remove as much fluid or blood as possible.

Arthrocentesis of the hip joint is generally performed by an orthopedic surgeon. It may be difficult to aspirate fluid from

small joints. If only one drop of fluid is obtained from small joints, it is best to send it for culture.

## The common complications of procedure

- Iatrogenic infection
- Iatrogenic hemorrhage
- Pain during the time of the procedure
- Reaccumulation of the joint fluid

## Specific Arthrocentesis Techniques

Landmarks and positioning are important while performing arthrocentesis. For small joints, application of traction is often very helpful in obtaining fluid.

### Radiohumeral Joint (Elbow)

#### Lateral approach

- The patient sits upright on a stretcher.
- Bend the patient's elbow to 90°.

- Pronate the patient's forearm and rest it with the palm down on a side table set at the appropriate height for comfort.
- Identify the olecranon process, lateral epicondyle, and radial head, and find the depression (or bulge, if the effusion is large) in the soft triangle. This site is used for all approaches.
- Identify the entry site, and mark the site with a plastic needle sheath or a sterile surgical marker.
- Carefully examine the elbow before arthrocentesis.
- Olecranon bursitis is located posteriorly over the olecranon and can be confused with the elbow joint.

The alternative is the posterolateral approach can be used. However, there is an increased risk of injury to the radial nerve and triceps tendon. This approach is useful if the bulge of effusion is palpated inferior to the lateral epicondyle. In the posterolateral approach, insert the needle perpendicular to the skin but

parallel to the radial shaft. The landmarks can be found easily if the arm is first extended. At this point, the depression can be located. Then flex and pronate the arm for the procedure.

Because of the risk of ulnar nerve and superior ulnar collateral artery injury, the medial approach should not be used.

How to locate the entry site; please watch the [video](#).

Real patient example (watch the [video](#))

### Radiocarpal Joint (Wrist)

The wrist joint is anatomically complex. The dorsal site is the preferred site of aspiration of the wrist joint.

The landmark of this joint is the dorsal radial tubercle (Lister's tubercle). The extensor pollicis longus tendon runs in a groove on the radial side of the tubercle. The tendon can be palpated by active extension of the wrist and thumb.

- The wrist should be slightly palmar flexed to facilitate the performance of



the procedure.

The positioning of the wrist is approximately 20 to 30 degrees of flexion with accompanying ulnar deviation.

- Applying traction to the hand might be helpful.
- Insert the needle dorsally just distal to the radius and just ulnar to the anatomic snuff box.
- Avoid the associated tendons (extensor carpi radialis brevis and extensor pollicis longus).
- Direct the needle perpendicular to the skin.
- If the bone is hit, pull the needle back and redirect it slightly toward the thumb.

Watch the [video 1](#) and [video 2](#).

## Glenohumeral Joint (Shoulder), Anterior Approach

- First of all arthrocentesis of this joint is moderately difficult.
- The patient should sit upright with the arm at the side, with the shoulder held in external rotation.
- To find the landmark clinician should palpate the coracoid process medially and the proximal end of the humerus laterally.
- The clinician should insert a 20-gauge needle at a point inferior and lateral to the coracoid process and direct it posteriorly toward the glenoid rim.

The [video](#) shows posterior approach.

## Knee Joint, Anteromedial Approach

The medial surface of the patella at the middle or superior portion of the patella is the landmark for the knee joint. Knee arthrocentesis may be done via the parapatellar approach (which is generally preferred), suprapatellar approach, or infrapatellar approach.

For the parapatellar approach, identify the midpoint of either the medial or the lateral border of the patella. Insert an 18-gauge needle 3-4 mm below the midpoint of either the medial or the lateral border of the patella. Direct the needle toward the intercondylar notch of the femur by perpendicular to its' long axis.

For the suprapatellar approach, identify the midpoint of either side of the superomedial or the superolateral border of the patella. Insert an 18-gauge needle through the midpoint of either superior borders. Direct the needle toward the intercondylar notch of the femur. The needle enters the suprapatellar bursa. Remember that in 10% of the population, the suprapatellar bursa does not communicate with the knee joint.

For the infrapatellar approach, position the patient sitting upright with the knee bent at 90° over the edge of the bed. Identify either side of the inferior border of the patella and the patellar tendon. Insert an 18-gauge needle 5 mm below

the inferior border of the patella and just lateral to the edge of the patellar tendon. Be careful not to go through the patellar tendon while inserting the needle.

Please watch the [video 1](#) and [video 2](#).

### **Tibiotalar Joint (Ankle)**

The medial malleolar sulcus is bordered medially by the medial malleolus and laterally by the anterior tibial tendon. The tendon can easily be identified with active dorsiflexion of the foot. The clinician should insert the needle at a point just medial to the anterior tibial tendon and directed into the hollow at the anterior edge of the medial malleolus. The needle must be inserted 2 to 3 cm to penetrate the joint space.

Please watch the [video](#).

### **Metatarsophalangeal and Interphalangeal Joints**

For the first digit, landmarks are the distal metatarsal head and the proximal base of the first phalanx. For the other toes, the landmarks are the prominences at the

proximal interphalangeal and distal interphalangeal joints. The extensor tendon of the great toe can be located by active extension of the toe. The clinician should insert the needle into the skin at a 90-degree angle and enter the dorsomedial aspect of the great toe (MTP) joint, just medial to the extensor tendon.

Please watch [video](#).

**References and Further Reading**, click [here](#)

# Basics of Bleeding Control

by Ana Spehonja and Gregor Prosen

## Types of wounds

### Contusion (Contusio)

It is a result of minor forces, usually over clothes on 90° angle. Capillaries beneath the skin can rupture due to a blunt blow or punch. There can also be a hematoma. Analgesics and RICE (rest, ice, compression, and elevation) will be enough for management.

### Abrasion (Excoriation)

It is a result of forces obliquely hit skin. The topmost layers of skin are scraped off, leaving a raw, tender area. Clean the wound, put a sterile bandage, give an analgesic, tetanus protection, and RICE (rest, ice, compression, and elevation) are the parts of management.

If the wound is deep, it may need a surgical consult or referral for better cosmetic healing. Please check the wound care chapter.

### Wound (Vulnus)

- caused by sharp object – vulnus scissum
- laceration – vulnus lacerum
- puncture wound – vulnus ictum
- bite wound – vulnus morsum
- gunshot wound – vulnus sclopetarium
- explosive wound – vulnus explosivum
- contusion – conquassatio

Types are based on time, place, the cause of injury. A clean wound which is not older than 6-8 hours (18-24 hours on face) can be closed right away. Wounds that are older than 8 hours should be thoroughly cleaned and cover with wet gauze. They can be closed after 3-5 days when they are clean, and there is no sign of infection.

If blood vessels affected,

## Arterial bleeding

It is a consequence of injury to the artery. The blood is pulsating out of the wound and has a bright red color. If the artery is lacerated through the whole lumen, it will spontaneously shrink and limit the bleeding. However, if there is only injury to the wall of an artery and it is not thoroughly dissected, this cause even more harm.

How to recognize arterial bleeding

## Hard Signs

- No pulses

- Bruit or thrill
- Active or pulsatile bleeding
- Signs of limb ischemia
- Pulsatile or expanding hematoma

## Soft Signs

- Proximity of injury to vascular structures
- Major single nerve deficit
- Non-expanding hematoma
- Reduced pulses
- Posterior knee or anterior elbow dislocation
- Hypotension or moderate blood loss at the scene

## Venous bleeding

It is a consequence of injury to the vein. The blood is leaving the wound more slowly and is not pulsating; it has a dark red color.

## Capillary bleeding

Damaged subcutaneous capillaries.  
Slow dotted bleeding.

## Assessment and Simple Procedures

### Initial evaluation when assessing wounds that are not life- or limb-threatening:

- past medical history and circumstances surrounding the injury,
- remove rings or other jewelry that encircle the injured body part,
- review the mechanism of injury,
- ask about the presence of a foreign body sensation,
- determine the time that the injury occurred
- determine if the wound was the result of intentional, unintentional or workplace event
- examine nerves' motor and sensorial function, and tendons.



## Assessment of bleeding wounds that are potentially life- or limb-threatening

### Direct pressure

We should provide equal pressure over a gauze that covers the complete wound. It is the first step of immediate bleeding control, and applicable anywhere on the body. Replace skin flaps to their original position, before applying pressure if possible.

Some areas of the body can be painful, and it is a limitation for some patients. Direct pressure has time limitations. Therefore, application of pressure bandage may be necessary.

### Pressure on arteries

We can stop blood flow to extremities with pressure on main arteries. It is the second step of immediate bleeding control.

It is useful only on extremities. It is a painful application and has time limitation as direct pressure.

## Eschmarch tourniquet

We can use cuff from blood pressure monitor. It should be inflated with pressure over 250mmHg, especially on lower extremities. However, inflating 20-30 mmHg over the systolic blood pressure levels are also acceptable in most of the bleeding. Use only to stop life-threatening exsanguination or when a tourniquet is needed for a short period of time to create a bloodless field for wound inspection. This technique can be used if above measures are not effective to stop a fast bleeding. It has a time limit up to 2 hours. It is a painful procedure. Apply blood pressure cuff proximal to the bleeding point, inflate it above systolic blood pressure and clamp the tubing with a hemostat. After procedure record the time of application, do a neurological exam and do not leave the tourniquet on for more than 120 minutes.

## Compression bandage

It is very fast, but a temporary bleeding control maneuver. It is less effective than

Eschmarch tourniquet. It can limit breathing when applied on the thorax. Lift the injured limb. Place clean gauze over the wound and maintain direct pressure on the wound. Place one bandage over the wound and wrap the other on around the limb. Make sure to have firm and constant pressure. Place the limb in a brace and keep it elevated. Check pulse, mobility, and sensation distal from the dressing. Check the dressing every 5-10 minutes.

## Clamping and Cauterisation

It is fast and on point bleeding control. One of the final steps, if the above measures do not work to stop bleeding. It should not be applied any wound and vessel having an amputation and possible re-anastomosis chance. But it is ideal for continuously bleeding superficial arteries in some wounds. Do not try to clamp deeper vessels because clamping may damage other structures.

Cauterization is a final step of bleeding control in the ED, and applied by surgical

teams when the other measures used by ER team are not effective to stop bleeding.

**References and Further Reading,** click [here](#)

# Cardiac Monitoring

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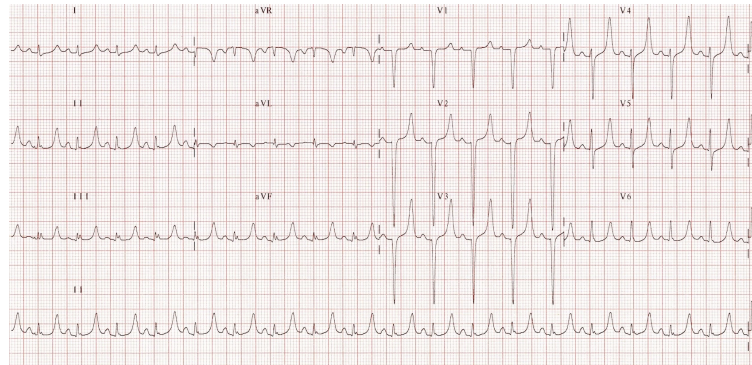
by Stacey Chamberlain

## Case Presentation

*A 44-year-old male patient with a history of hypertension and end-stage renal disease on hemodialysis presents with shortness of breath after missing dialysis for 6 days. He reported gradual onset shortness of breath associated with orthopnea and increased lower extremity edema. He denies chest pain or palpitations. He does not have any cough or fever. On physical exam, he is in no distress, afebrile with a heart rate of 60, respiratory rate of 20, blood pressure of 140/78 and oxygen saturation of 98% on room air. He has a regular rate and rhythm without murmurs and has crackles bilaterally to the inferior 1/3 of the lung bases and 1+ pitting edema of the bilateral lower extremities.*

*You decide to get an EKG which shows the following (EKG from [www.lifeinthefastlane.com](http://www.lifeinthefastlane.com)):*

**Image 16.4 ECG 1**



<https://i2.wp.com/lifeinthefastlane.com/wp-content/uploads/2011/02/ECG-Potassium-7-peaked-T-waves.jpg?ssl=1>

*You send a blood chemistry test, place the patient on a cardiac monitor and one hour later note the following on the monitor (EKG from [www.lifeinthefastlane.com](http://www.lifeinthefastlane.com)):*

**Image 16.5 ECG 2**



<https://i0.wp.com/lifeinthefastlane.com/wp-content/uploads/2011/02/disappearance-p-waves-hyperk.jpg?ssl=1>

*What are the indications for cardiac monitoring in this patient? What EKG abnormalities do you see? What does the rhythm strip show? What is the treatment?*

*Case discussion is at the end of the chapter.*

## Introduction

Cardiac monitoring in the emergency setting is continuous monitoring of a patient's cardiac activity in order to identify conditions that may require emergent intervention. These conditions include certain arrhythmias, ischemia and infarction, and abnormal findings that could signal impending decompensation. This chapter focuses specifically on cardiac monitoring or electrocardiography.

Additional methods of continuous hemodynamic monitoring in the ED

include pulse oximetry, end tidal CO<sub>2</sub> monitoring, central venous pressure monitoring, and continuous arterial blood pressure monitoring. Of note, telemetry is the ability to do cardiac monitoring from a remote location; in practice, this is often a centralized system that might be located at a nursing station where multiple patients can be monitored remotely.

Cardiac monitoring differs from a 12-lead electrocardiogram in that it is done continuously over a period of time rather than capturing one moment in time in a static image. The benefit of this, of course, is for capturing transient arrhythmias, ectopic beats, or monitoring for changes over time. A disadvantage of cardiac monitoring is that typically only 2 leads are displayed instead of a full 12 leads, giving a less comprehensive view of the heart and limiting its utility to look for anatomic patterns. For example, on the 12 lead EKG, ED practitioners usually group the inferior, anterior and lateral leads when looking for ischemic or infarct patterns. These may be less evident on a



monitor with only two leads. Additionally, the static EKG allows for the ED physician to carefully study it for subtle findings, for example, to make measurements of intervals, whereas, in real-time monitoring, this is very difficult. In practice, both modalities are commonly used in conjunction for many ED patients.

The American Heart Association (AHA) published a consensus document in 2004 establishing practice standards for electrocardiographic monitoring in hospital settings. This comprehensive document outlines the indications for cardiac monitoring, the specific skills required of the practitioner for cardiac monitoring, and specific ECG abnormalities that the practitioner should recognize.

Cardiac monitoring is essential for those patients who are at risk for an acute, life-threatening arrhythmia. The AHA guidelines divide indications for cardiac monitoring in the inpatient setting into

three classes. Cardiac monitoring is considered indicated in “most, if not all” patients in Class I, which includes 16 subcategories. In Class II, cardiac monitoring “may be of benefit in some patients but is not considered essential for all patients” and has 10 subcategories. For Class III, cardiac monitoring is not indicated.

## Indications for Cardiac Monitoring

Adopted from AHA consensus document

### Class I Indications

Cardiac monitoring is considered indicated in “most, if not all” patients in Class I

1. Patients who have been resuscitated from cardiac arrest
2. Patients in the early phase of acute coronary syndromes (ST-elevation or non-ST-elevation MI, unstable angina/“rule-out” MI)

3. Patients with unstable coronary syndromes and newly diagnosed high-risk coronary lesions (for 24 hours)
4. Adults or children who have undergone cardiac surgery (minimum of 48 to 72 hours)
5. Patients who have undergone non-urgent percutaneous coronary intervention with complications
6. Patients who have undergone implantation of an automatic defibrillator lead or a pacemaker lead and are considered pacemaker dependent
7. Patients with a temporary pacemaker or transcutaneous pacing pads
8. Patients with AV block
9. Patients with arrhythmias complicating Wolff-Parkinson-White syndrome with rapid anterograde conduction over an accessory pathway

10. Patients with long-QT syndrome and associated ventricular arrhythmias
11. Patients receiving intra-aortic balloon counter-pulsation
12. Patients with acute heart failure/ pulmonary edema
13. Patients with indications for intensive care
14. Patients undergoing diagnostic/ therapeutic procedures requiring conscious sedation or anesthesia
15. Patients with any other hemodynamically unstable arrhythmia
16. Diagnosis of arrhythmias in pediatric patients

### **Class II Indications**

Cardiac monitoring “may be of benefit in some patients but is not considered essential for all patients.”

1. Patients with post-acute MI (24 to 48 hours after admission)

2. Patients with chest pain syndromes
3. Patients who have undergone uncomplicated, non-urgent percutaneous coronary interventions
4. Patients who are administered an antiarrhythmic drug or who require adjustment of drugs for rate control with chronic atrial tachyarrhythmias
5. Patients who have undergone implantation of a pacemaker lead and are not pacemaker dependent
6. Patients who have undergone uncomplicated ablation of an arrhythmia
7. Patients who have undergone routine coronary angiography
8. Patients with sub-acute heart failure
9. Patients who are being evaluated for syncope
10. Patients with do-not-resuscitate orders with arrhythmias that cause discomfort

### **Class III**

Cardiac monitoring is not indicated

1. Postoperative patients who are at low risk for cardiac arrhythmias (e.g. young patients without heart disease who undergo uncomplicated surgical procedures)
2. Obstetric patients, unless heart disease is present
3. Patients with permanent, rate-controlled atrial fibrillation
4. Patients undergoing hemodialysis (unless they have a class I or II indication)
5. Stable patients with chronic ventricular premature beats

### **“Must Know” Arrhythmias**

One of the most critical skills of an ED physician is interpreting both static EKGs and interpreting arrhythmias on a cardiac monitor. A skilled practitioner must be able to diagnose common arrhythmias and be well versed in the management of

acute arrhythmias, recognizing which arrhythmias necessitate immediate action and which are less worrisome. AHA guidelines list the specific arrhythmias that the ED physician must be able to recognize.

### Specific Arrhythmias (adopted from AHA Scientific Statement)

- Normal rhythms
  - Normal sinus rhythm
  - Sinus bradycardia
  - Sinus arrhythmia
  - Sinus tachycardia
- Intraventricular conduction defects
  - Right and left bundle-branch block
  - Aberrant ventricular conduction
- Bradyarrhythmias
  - Inappropriate sinus bradycardia
  - Sinus node pause or arrest
- AV blocks
  - 1st-degree
  - 2nd-degree Mobitz I (Wenckebach) or Mobitz II
  - 3rd-degree (complete heart block)
- Asystole
- Pulseless electrical activity (PEA)
- Tachyarrhythmias
  - Supraventricular
    - Non-conducted atrial premature beats
    - Junctional rhythm
    - Junctional ectopic tachycardia
    - Accelerated ventricular rhythm
  - Paroxysmal supraventricular tachycardia (AV nodal reentrant, AV reentrant)
  - Atrial fibrillation
  - Atrial flutter
  - Multifocal atrial tachycardia

- Ventricular
  - Monomorphic and polymorphic ventricular tachycardia
  - Torsades de pointes
  - Ventricular fibrillation
- Premature complexes
  - Supraventricular (atrial, junctional)
  - Ventricular
- Muscle or other artifacts simulating arrhythmias

### Approach

How and whether to treat an arrhythmia depends on many factors. The AHA has established algorithms for specific rhythms including ventricular fibrillation (v-fib)/pulseless ventricular tachycardia (v-tach) and pulseless electrical activity (PEA)/asystole, as well as for non-specific rhythm categories such as bradycardia

and tachycardia. Additionally, they have published algorithms for clinical scenarios including cardiac arrest, acute coronary syndrome, and suspected stroke.

The first step in the assessment of any rhythm is a clinical assessment of the patient. The premier issue of concern is if the patient is perfusing vital organs. A quick survey of the patient assessing mental status and pulses is essential to determining management. The management of a patient with v-tach will be substantially different if the patient is unresponsive and pulseless versus if the patient is awake with good pulses. As another example, the physician can quickly distinguish artifact from v-fib on the cardiac monitor by assessing the patient, as v-fib is not a perfusing rhythm.

The initial assessment of tachyarrhythmias (heart rate > 100) is to determine if the rhythm is “narrow-complex” (i.e., QRS duration < 0.12s) or “wide-complex” (i.e., a QRS duration of 0.12s or greater). A narrow complex

rhythm is considered a supraventricular rhythm (originating above the ventricles). Supraventricular tachycardia is a generic term encompassing any narrow-complex tachycardias originating from above the AV node. Colloquially, when many practitioners refer to “SVT” however, they are actually referring to a specific subcategory of supraventricular tachycardia called AV nodal re-entrant tachycardia (AVNRT). Wide complex tachycardias either originate in the ventricles or could originate in the atria and have an associated bundle branch block. Different criteria have been developed to help the practitioner distinguish between ventricular tachycardia and an SVT “with aberrancy” (i.e., aberrant conduction either due to an accessory path such as in Wolff-Parkinson-White or with a bundle branch block), the most well known of which are the Brugada criteria. Practically speaking, many ED practitioners will assume the more dangerous and potentially unstable rhythm (v-tach) until proven otherwise; of course, the clinical

picture and patient’s vital signs are of utmost importance in determining the management for these patients. A nice summary of this issue with rhythm strip examples is provided on the FOAM site “Life in the Fast Lane.”

While each rhythm has distinctive management, it is worth noting for the novice learner that only v-fib and pulseless v-tach warrant asynchronized mechanical defibrillation (i.e., “shocking” the patient). Many students are stunned upon observing an asystolic cardiac arrest code to learn that shocking a “flatline” (i.e., asystolic) patient is an inappropriate treatment perpetrated by fictitious TV shows and movies. For unstable patients with arrhythmias in patients who still have palpable pulses, synchronized cardioversion may be used.

In regards to medications, for certain rhythms and clinical scenarios, only vasopressor types of medications are used (e.g., epinephrine for asystole). For other rhythms and scenarios, anti-



arrhythmic medications are used (e.g., amiodarone for v-tach). For supraventricular tachyarrhythmias, atrioventricular (AV) nodal blocking agents are often necessary. One author suggests using a five “As” approach to treating emergency arrhythmias, keeping in mind the medications adenosine, amiodarone, adrenaline (epinephrine), atropine and ajmaline. Ajmaline is an antiarrhythmic that is not commonly used in English-speaking countries where procainamide is more common as an alternative to amiodarone for unstable v-tach.

Additional interventions may include pacemaker placement for symptomatic heart blocks and in many cases, determining the underlying precipitant of the arrhythmia and tailoring treatment to that cause. The emergency physician must familiarize himself with each rhythm and its unique management in any given clinical scenario.

Some useful internet resources for the ED practitioner are provided at the end of this chapter for practice interpreting EKGs and cardiac rhythms.

## Case Discussion

The ED practitioner should recognize that the potentially life-threatening conditions that a patient who has missed hemodialysis is at risk for are fluid overload (leading to pulmonary edema) and hyperkalemia. This patient could be considered to meet the Class I monitoring criteria for “needing intensive care” and possibly with “pulmonary edema;” however, even if the patient had no symptoms, the patient is at risk for an acute life-threatening arrhythmia that would necessitate cardiac monitoring.

The EKG demonstrates peaked T waves indicative of acute hyperkalemia. Given the clinical picture of missed dialysis and the peaked Ts on the EKG, the ED physician should immediately initiate treatment for acute hyperkalemia without waiting for a confirmatory blood test

(unless an immediate point of care tests are available). If the patient’s hyperkalemia progressed, the patient could develop QRS widening with the morphology as shown on the rhythm strip called a “sine wave.” This dangerous finding could precipitously deteriorate into a life-threatening arrhythmia such as pulseless v-tach with cardiac arrest and should prompt immediate action. It is important to note that hyperkalemia can manifest in a variety of different EKG findings and does not always follow a consistent pattern from peaked Ts to QRS widening to sine waves; therefore, the patient should be treated at the first indication of any hyperkalemia-related EKG changes.

**References and Further Reading,** click [here](#)

# Gastric Lavage and Activated Charcoal Application

---

by Elif Dilek Cakal

## Case Presentation

*A 22-year old female presented to the emergency department 15 minutes after she had committed suicide by taking 30 pills of 500 mg acetaminophen. She had no known chronic diseases. Her blood pressure was 134/87 mmHg; temperature, 36.4°C; heart rate of 70 bpm and regular; respiration 15 bpm; and O2 saturation 99%. At the time of arrival, she was asymptomatic. Nothing was remarkable on examination. Gastric lavage was performed. 1 mg/kg of activated charcoal was given to the patient. IV N-acetylcysteine treatment was started. She was admitted to the hospital.*

## Gastric Lavage Procedure

### Emergency Indications

Gastric Lavage (GL) should not be undertaken routinely. Whether gastric lavage positively alters the morbidity or mortality of the poisoned patient, even applied shortly after the intake, is controversial. GL is indicated only if:

- Oral intake < 60 minutes
- The life-threatening dose of the toxic substance is ingested

### Contraindications

- Patients with compromised airway reflexes, unless they are intubated. If the critical situation of the patient indicates intubation, then, gastric lavage may be performed. Intubation, only for decontamination, is not recommended.
- Non-toxic or non-life-threatening intoxications.

- Hydrocarbons intake (unless containing highly toxic substances such as pesticides).
- Oral intake of caustic substances.
- Poisonings with toxic substances; those are more toxic to lungs than to gastrointestinal system.
- Poisonings with pills that are known not to fit through the holes of the gastric tube
- Known esophageal structures.
- History of gastric bypass surgery.

Emergency Physician (EP) must be cautious in combative patients and patients with medical conditions such as bleeding disorders.

## Equipment and Patient Preparation

### Equipment for GL includes:

- Intravenous access and monitoring
- A large suction catheter

• Local analgesics and lubricants

- Intubation equipment
- Sedatives (if necessary)
- Restraints (if necessary)
- Bite block or oral airway
- Oral or nasogastric tubes
  - 36- to 40-French or 30 English-gauge tubes in adults (oral)
  - 24- to 28- French-gauge in children (oral)
- Lavage systems
  - Commercially available
  - Intermittent aliquots of lavage fluid can be given and withdrawn manually

• Activated charcoal (see below)

• Normal saline or water

Before starting, the steps of the procedure must be explained to patients

in an attempt to gain cooperation. If the patient is too agitated, sedatives in anxiolytic doses may be used. EP must keep in mind that significantly altered level of consciousness due to sedation warrants intubation.

Although there is no adequate data in humans to show that tube diameter or route is important, the oral route is primarily preferred for the gastric lavage. Nasogastric tubes are less traumatic for patients and are preferred in liquid ingestions and children.

Place all patients in the left lateral decubitus position in Trendelenburg to facilitate the content removal and to decrease the aspiration risk. Supine position greatly increases aspiration risk, unless the patient is intubated.

The tube must be measured from the corner of the mouth to the mid-epigastrium in order to avoid kinking and complications.

## Procedure Steps

This [video](#) explains the steps of the insertion of the gastric tube

1. Explain the procedure to the patient.
2. Collect the equipment and place the patient in the left lateral decubitus position.
3. Put a bite block or oral airway into the patient's mouth.
4. Introduce to pass the tube gently
5. When the pharynx is reached, put the patient's chin on the chest to facilitate passage of the tube into the esophagus.
6. Confirm the placement
7. Aspirate and remove the gastric contents before gastric irrigation
8. Repeatedly introduce 200–300 mL of lavage solution (10 mL/kg body weight in children up to a maximum of 300 mL) into the stomach and then remove them

9. Continue lavage until the fluid becomes clear

10. Administer activated charcoal via tube

11. Clamp off and remove the tube

## Hints and Pitfalls

- The procedure is intended to be therapeutic, not punitive.
- In some situations, Gastric lavage may be helpful for up to 2 hours:
  - Highly toxic drugs
  - Drugs not absorbed by activated charcoal
  - Sustained release or enteric-coated products
- Auscultation of the stomach generally confirms the placement of the tube during injection of air with a 50-mL syringe and aspiration of gastric contents. Radiographic confirmation should be considered, especially in children and intubated patients.



- A cough, stridor, or cyanosis indicates that the tube has entered the trachea; withdraw the tube immediately and reattempt passage.

## Post Procedure Care and Recommendations

- For most patients, a short period of observation of vital signs is adequate.
- The nature of the poisoning will lead the management.

## Complications

GL is generally safe but not harmless.

Complications of GL include:

- Misplacement of the gastric tube into the trachea.
- Pulmonary aspiration of gastric content of lavage fluid, especially in patients with compromised airway reflexes.
- Aspiration pneumonia.
- Laryngospasm and hypoxia, especially in patients with lung diseases.

- Esophageal lacerations or perforation.
- Gastric perforation.
- Fluid and electrolyte disturbances, especially in children.
- Hypothermia.
- Nasal, oral, pharyngeal, pyriform sinus injuries.
- Pulmonary hemorrhage, pneumothorax, and empyema.

## Pediatric, Geriatric, and Pregnant Patient Considerations

Gastric lavage is always a difficult procedure to apply to children. Nasogastric tubes may be preferred. 10 mL/kg aliquots of lavage solution up to a maximum of 300 mL is given and removed. Because electrolyte disturbance has occurred in children who were lavage with tap water, prewarmed (45°C) normal saline is generally recommended for children.

Elderly patients are susceptible to cardiac consequences of both procedure and the poisoning; therefore, their vital signs should be monitored closely.

Gastric lavage and activated charcoal are considered safe for pregnant patients. Poisonings that are toxic to the fetus as well as toxic to mother must be considered.

## Activated Charcoal Application

### Emergency Indications

- Oral intake < 60 minutes
- the life-threatening dose of the toxic substance

### Multi-Dose Activated Charcoal (MDAC) Indications

Life-Threatening Oral Intake of

- Carbamazepine
- Dapsone
- Phenobarbital

- Quinine
- Theophylline

## Contraindications

- For patients with compromised airway reflexes, unless they are intubated. If the critical situation of the patient indicates intubation, then, gastric lavage may be performed. Intubation, only for decontamination, is not recommended.
- Oral intake of caustic substances
- Late presentation
- Increased risk and severity of aspiration associated with AC use (e.g., hydrocarbon ingestion)
- Need for endoscopy (e.g., significant caustic ingestion)
- Toxins poorly adsorbed by AC (e.g., metals including iron and lithium, alkali, mineral acids, alcohols)
- Presence of intestinal obstruction (absolute contraindication) or concern

for decreased peristalsis (relative contraindication)

## Equipment and Patient Preparation

There is no specific equipment for activated charcoal administration. However, drinking the charcoal can be very unpleasant for many patients, especially children. Therefore, mixing with fruit juice can be an option. In addition, if necessary nasogastric or orogastric tube placement can facilitate the active charcoal treatment.

## Procedure Steps

Recommended empirical single-dose of activated charcoal is as follows:

- <1 year – 0.5-1 g/kg or 10-25 g
- 1-12 years – 0.5-1 g/kg or 25-50 g
- >12 years – 1-2 g/kg or 25-100 g

Multidose activated charcoal

• Give the recurrent dose of charcoal by 0.5 g/kg ( $\leq 50$  g) every 4 hours

How to administer:

- If the patient is awake and cooperative, AC may be given orally. Alternatively, it may be given by gastric or nasogastric tube, if these procedures are indicated.
- Mixing the activated charcoal with fruit juices increases tolerability.
- If the patient is unconscious or airway is compromised, gastric lavage should be done, and activated charcoal should be given after intubation. Tracheal intubation is not recommended solely in order to give activated charcoal. Only activated charcoal is to be given, the nasogastric tube is adequate and is preferred.
- If MDAC is indicated, the gastric tube should be withdrawn after gastric lavage and the first dose of activated charcoal. Further doses should be given via nasogastric tube.

## Hints and Pitfalls

- The substances that cannot bind to activated charcoal are as follows:
  - Lithium
  - Strong acids and bases
  - Metals and inorganic minerals
  - Alcohols
  - Hydrocarbons
- Multi-dose activated charcoal enhances elimination of (But not necessarily indicated in all)
  - Amitriptyline
  - Aspirin
  - Caffeine
  - Carbamazepine
  - Cyclosporine
  - Dapsone
  - Digoxin

- Disopyramide
- Nadolol
- Phenobarbital
- Phenytoin
- Piroxicam
- Quinine
- Sotalol
- Sustained-release thallium
- Theophylline
- Valproate
- Vancomycin

- MDAC increase the risk of constipation and bowel obstruction in some cases. Therefore, consider adding a cathartic agent to the second or third dose of AC.

## Post Procedure Care and Recommendations

- Control possible nausea and vomiting.

- Look for traces of aspiration or gastrointestinal complications.

## Complications

Complications of AC and MDAC include:

- Constipation, diarrhea, vomiting
- Pulmonary aspiration

## Pediatric, Geriatric, and Pregnant Patient Considerations

In pediatric and geriatric patients, extra caution should be exercised to avoid and monitor complications.

Activated charcoal is considered safe for pregnant women.

**References and Further Reading**, click [here](#)

# Intravenous (IV) Line Access

---

by Keith A. Raymond

## Introduction

Peripheral Intravenous (IV) cannulation is a nursing skill. Few countries throughout the world require physicians to perform this procedure on a regular basis. Mastery of technique, understanding nuances and anatomy, and daily performance are required to maintain this skill. Therefore, if a nurse reports that he is unable to obtain IV access, and it is required urgently, establishing an IV access or intraosseous (IO) line should be considered to avoid delay.

IV lines can safely remain in place safely for up to 72 hours. In some cases, this is up to 7 days.

*“There is no body cavity that cannot be reached with a number fourteen needle and a good strong arm.”*

*— Samuel Shem, The House of God*

## IV Line Access and Procedure

Success rates in multiple attempts for admitted patients at a children’s hospital range from 23% for physicians, 44% for nurses to 98% for IV nurse clinicians. The average time required for peripheral IV cannulation is reported at 2.5 to 13 minutes, with difficult IV access requiring as much as 30 minutes. Therefore, we will focus here on peripheral IV cannulation and line access for the easiest and



most commonly used sites in emergencies, as we must provide high volumes and medications to the patient quickly.

### Emergency Indications

Intravenous access is used when therapies cannot be used or are less effective by alternative routes. In critical situations, medication bioavailability, hydration, and blood products can be given and provide rapid onset of action. Peripheral access is typically safer, easier to obtain, and less painful than central access. Finally, two large bore intravenous catheters in place can provide the same or more fluids during resuscitation as a central line.

### Contraindications

- Patients with anatomic disparities that could lead to fluid or medication extravasation locally or proximally.
- Massive edema in extremities, burns, cellulitis, or injuries at or proximal to proposed insertion sites.

- Any site where there is a concern for vascular flow.

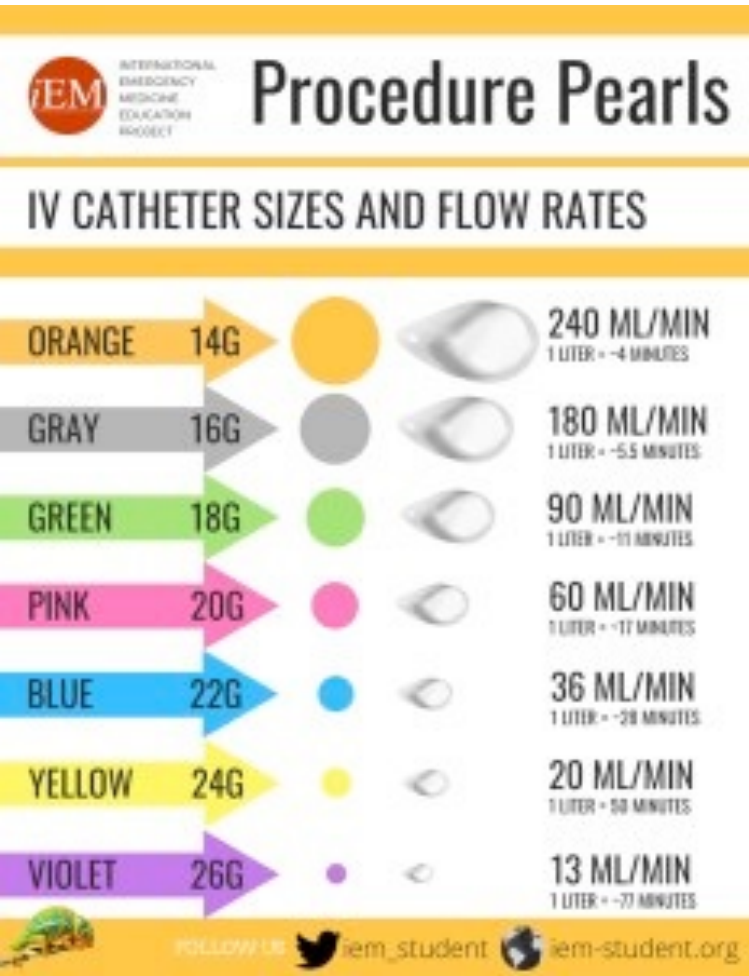
### Equipment and Patient Preparation

#### Equipment

- gloves,
- skin disinfectant (Povidine and Alcohol Swabs),
- 16-18 gauge IV catheter (smaller catheters may be used for pediatric patients, but larger is better in critical cases), (see Illustration 16.1)
- tape,
- syringe,
- 3-way stopcock,
- isotonic crystalloid solution,
- intravenous tubing,
- an elastic tourniquet or blood pressure (BP) cuff.
- Optional

- Topical anesthetic, eg. EMLA (2.5% lidocaine and prilocaine),
- transilluminator light,
- ultrasound with a vascular probe.

Illustration 16.1



## Patient Preparation

- Obtain informed consent or implied, following procedure discussion, risks, and benefits.
- If possible, have the patients wash their forearms, including the antecubital space, three times with soap and water, then pat dry.
- Select the site starting distally, preferred Cephalic vein in the forearm, then Medial Brachial Vein in Antecubital Sulcus.
- Always apply universal precautions (gloves as a minimum) to the procedural list. Both visualize and palpate the vein to be cannulated.
- There is a slight give to the vessel compared to surrounding tissue.
- Disinfect overlying skin, and provide topical anesthetic to site as desired.
- Transillumination and/or ultrasound may be used to provide additional guidance,

but prevent contamination of the clean prepped site to be accessed.

## Procedure

1. Apply the tourniquet or BP cuff (inflate above diastolic reading) proximal to the intravenous site.
2. Using 'no-touch' technique, insert the IV catheter distal to and along the line of the vein at a 10 to 15-degree angle to the skin.
3. Advance the needle and the catheter slowly; in most cases, a 'flash' of blood will enter the catheter (but not always).
4. SLOWLY advance the needle an additional 1 to 2 millimeters, then slide the cannula into the vein, while securing the needle in place.
5. Remove the needle while pressing on the overlying skin over the cannula proximal to the insertion site to stem the blood flow.

6. Attach the 3-way stopcock, then flush the stopcock and cannula of blood with 5 ml of saline to prevent clotting, and assess the flow of fluid through the catheter. Watch for skin bulge suggesting extravasation of fluid.

7. Secure the catheter with tape and release the tourniquet or BP cuff.
8. Attach intravenous tubing to 3 way stopcock, attached to the fluid of choice and initiate flow, watching again for fluid extravasation. Medications may be administered through another port of the stopcock or added to the IV solution as desired.
9. Make sure that you removed the tourniquet before you give drug or fluid infusion.
10. If fluid extravasation occurs at any time, remove the catheter, and repeat the procedure at the more proximal site (never distal to the previous attempt).

Please watch the [video](#).

## Post Procedure Care

- All medications administered should be followed by a 20 ml saline flush.
- A three-way stopcock should remain attached to the IV line if it is not in active use.
- Clean surrounding skin of blood and other contaminants following insertion.
- All IV catheters should be removed within 7 days or as soon as no longer necessary.
- Be vigilant during infusions for tissue swelling or catheter displacement.

## Hints and Pitfalls

- Palpation is more important than visualization.
- Secure vein proximally and distally from the insertion site if dealing with a 'roller' vessel.

- Use an arm board in pediatric patients, to prevent catheter displacement from movement.
- Do not use flashlights for transillumination as they can burn skin, use transilluminator only. Lowering the room light during transillumination maximizes visualization.
- Following two failed attempts, seek assistance and/or switch to an Intraosseousline.

## Complications

- Thrombosis and Hemorrhage
- Air embolism
- Extravasation of Drugs
- Vasculitis and Contusions

**References and Further Reading**, click [here](#)

# Intraosseous (IO) Line/Access

---

by Keith A. Raymond

## Introduction

Peripheral Intravenous (IV) cannulation is a nursing skill. Few countries throughout the world require physicians to perform this procedure on a regular basis. Mastery of technique, understanding nuances and anatomy, and daily performance are required to maintain this skill. Therefore, if a nurse reports that he is unable to obtain IV access, and it is required urgently, establishing an IV access or intraosseous (IO) line should be considered to avoid delay.

Following medicine delivery and fluid resuscitation utilizing an IO line, transition to peripheral intravenous or central intravenous access is easier to achieve, and the intraosseous line may be discontinued.

Intraosseous lines can safely remain in place for up to 24 hours and are often a bridge to either IV or Central Venous line placement.

## Intraosseous Line Access and Procedure

### Emergency Indications

When IV access cannot be achieved, IO access is safe, reliable, and quick. It can be accomplished in 30 to 60 seconds and even faster with an IO gun. This is especially helpful in pediatric emergencies when time is critical. Almost anything that can be given IV such as medications, fluids, blood products and continuous



infusions of catecholamines (epinephrine, norepinephrine, and dopamine).

## Contraindications

Absolute:

- fracture or crush injuries near or proximal to the access site,
- fragile bone conditions such as Osteogenesis Imperfecta,
- previous attempts in the same bone,
- the presence of infection in or on the overlying tissue of the bone,
- demineralized or immature bone.

Relative:

- IV access can be obtained readily.
- Use for only ultra short-acting medications such as Adenosine.

## Equipment and Patient Preparation

### Equipment

- gloves,

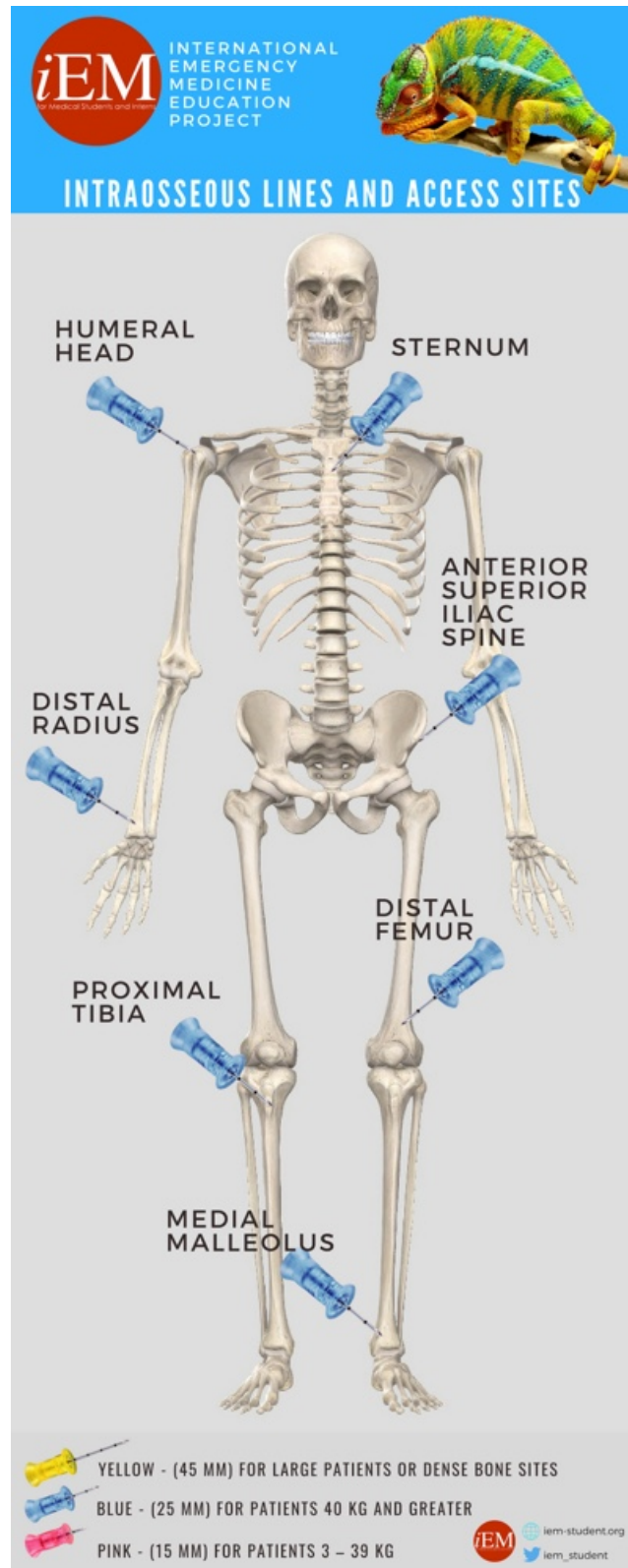
- skin disinfectant (Povidine or Chlorhexidine and Alcohol Swabs),
- 16-18 gauge IO or Jamshidi-type needle,
- tape,
- syringe,
- isotonic crystalloid solution, and intravenous tubing.
- Optional:
  - IO drill or gun, Infusion pump,
  - 2% Lidocaine for topical and subcutaneous infiltration (awake patients tend to report pain with fluid infusion rather than insertion).
- NOTE: Color coding of IO needles is common
  - Pink (15 mm) for patients 3 – 39 kg,
  - Blue (25 mm) for patients 40 kg and greater,

- Yellow (45 mm) for large patients or dense bone sites such as proximal humerus or anterior superior iliac spine.

## Patient Preparation

- Obtain informed consent or implied, following procedure discussion, risks, and benefits.
- Select site: humeral head, proximal tibia, medial malleolus, sternum, distal radius, distal femur, and/or anterior superior iliac spine. (see illustration 16.2)
- Proximal Tibia and Humeral Head are most commonly used during arrests as placement does not interfere with intubation or other activities.
- Always apply universal precautions (gloves as a minimum) to the procedural list.

## Illustration 16.2



## Procedure

1. Once the patient is prepared, identify the designated site with a sterile gloved finger.
2. Disinfect overlying skin, and provide local anesthetic as desired.
3. Be sure the stylet is in place on the needle prior to insertion.
4. Have a 20 ml Saline syringe flush, IV tubing, tape, medications, fluids, and pump prepared, as required.
5. Place the needle through the skin, perpendicular and down to the bone.
6. Activate the IO drill or gun until the IO needle anchors in place, OR manually TWIST the needle clockwise (don't push) with gentle firm pressure until the bone gives (loss of resistance technique) and the needle locks into place.
7. The bone give is an indication the needle has passed through cortical bone into the marrow.

8. If properly positioned, the needle will stand without support and be fixed in place.

- Remove the stylet and attach the syringe and aspirate, marrow and blood confirms placement but may not always appear.

9. Gently flush saline through the needle and watch the insertion site for swelling.

10. If the test injection is unsuccessful or swelling is seen on the opposite side of the bone, repeat the above procedure with a new IO needle on another bone.

11. If successful, stabilize the needle with the tape; gauze padding may be used as desired.

12. Attach the IV tubing to the needle hub and infuse fluids, blood products, or medications.

**Video** – Intraosseous Needle Line Insertion

**Video** – Intraosseous Needle Line Insertion in A Real Patient

## Post Procedure Care

- All medications administered should be followed by a 20 ml Saline flush.
- A three-way stopcock should be attached to the IO line if it is not in active use.
- All IO needles should be removed within 24 hours or as soon as an IV or Central line is placed.
- Be vigilant during infusions for tissue swelling or needle displacement.

## Hints and Pitfalls

- Always used an uninjured limb; if none available, the sternum is best.
- An IO drill or gun should be used preferentially to manual insertion
- In pediatric patients, if the bone is too soft, needle displacement is inevitable despite proper placement. Select anterior superior iliac spine.

- IO needle selection should be consistent with the site and marrow cavity.
- IO needle displacement sometimes can be avoided by properly securing it to the skin.

## Complications

- Bone fracture
- Compartment Syndrome
- Extravasation of Drugs
- Osteomyelitis

**References and Further Reading**, click [here](#)

# Emergency Delivery

---

by David F. Toro, Diana V. Yepes, Ryan H. Holzhauser

## Case Presentation

*As you begin the morning of your next weekend day shift in a small community hospital, the triage nurse comes in running and asks you to evaluate a patient that is being registered in the Emergency Department. You find a visibly pregnant 29-year-old female patient complaining of having regular uterine contractions for the last 10 hours and passed a significant amount of clear liquid per vagina on the way to the hospital, as well as a sensation of pelvic fullness and an increasing urge to use the bathroom with every uterine contraction.*

*On your evaluation, you find the patient is having uterine contractions at regular intervals, 4 times on a 10-minute period, lasting around 3 minutes each. You are able to detect a normal fetal heart rate and fetal movements. On the pelvic exam, you find a fully effaced and dilated cervix and palpate the fetal head at the level of the ischial spines. Only at this*



*moment, you remember your hospital does not have a gynecologist in-house, and your nearest transfer center is 1 hour away. What would you do next?*

## Introduction

Every year around 4 million babies are born in the US; unfortunately, there is no information on how many of these are born outside the regular delivery units, including the Emergency Department. Fortunately, however, it is an uncommon occurrence in Emergency Medicine. Just as it applies to many other emergency procedures, the Emergency Medicine provider needs to be familiar with the normal vaginal delivery. The provider must know preparations for it as well as how to identify and treat immediate complications for those cases where immediate access to an obstetrician is not readily available or if delivery is imminent before arrival to a birthing unit, such as in a patient arriving late to the hospital or a precipitous delivery.

This chapter describes the evaluation of the patient in possible active labor, the normal delivery technique, and immediate post-delivery care.

## Identifying True Labor

Labor is the process by which the fetus is expelled from the uterus and can be a lengthy process on nulliparous women but becomes a shorter process on subsequent pregnancies. It begins when an organized uterine activity starts, causing gradual effacement or thinning of the cervix and dilatation in order to allow passage of the fetus during the final stages.

The labor process can be divided into latent and active phases. The latent phase begins when there is organized and regular uterine activity causing a cervical dilatation and effacement; it is considered active phase when it causes 3 or more cm dilatation and/or effacement of 80%.

The active labor is normally divided into 4 stages. The first stage concludes when dilatation and effacement are complete. The second stage ends when the fetus is delivered, the third stage ends when the placenta is delivered, and the 4th stage is

the approximate period of 1 hour after the third stage concludes.

Contractions occur since the 2nd trimester as Braxton-Hicks contractions, but they become more common as the 3rd trimester goes by, transforming gradually into active labor. Braxton-Hicks contractions tend to be limited to the suprapubic area and thighs, are short and irregular in duration, have a low strength and are sporadic in timing.

True labor contractions, in contrast, are progressively longer in duration, radiated to the back and pelvic area, occur at regular intervals that become more frequent, are progressively stronger and cause effacement and dilatation of the cervix.

Other signs that indicate true labor are rupture of membranes and “bloody show.” The spontaneous rupture of membranes manifests by a sudden gush of clear fluid or by continuous leakage of vaginal fluid, with bleach or semen smell,

but may not occur until the moment of delivery.

Although Vaginal pH changes during pregnancy, normal vaginal fluid tends to have an acidic pH (4.5-6.0) where as amniotic fluid is alkaline (pH 7.0-7.5). Therefore, another way of identifying amniotic fluid is using nitrazine or pH paper. Under acidic environment, this paper changes color from yellow to orange, and when amniotic fluid is present, it changes from orange to yellow, green or blue (Image 16.6).

**Image 16.6** Under the presence of amniotic fluid, nitrazine paper turns from orange to yellow, green or blue.



“Bloody show” is the common name given to the expulsion of the blood-tinged cervical mucus plug as effacement and dilatation occur. Although by definition it is always present, in practice, it may not be noticed as it can occur gradually instead of all at once. When noticed, it can precede the initiation of active labor by several days.

## Initial Examination

In order to plan ahead for the imminent delivery, it is important to perform a vaginal and abdominal exam to determine the fetal well-being, lie, position, presentation, dilatation, effacement, and station.

## Abdominal exam

The Leopold maneuvers are part of the abdominal exam.

- First, palpate the uterine fundus to determine if the fetus is in a vertical or transverse lie by feeling if the fetal pole represents the head, breech (buttocks) or back.

- Second, apply pressure to the sides of the uterus with the entire hand, being sure to utilize both hands, to determine where the spine and extremities are.
- Third, with your dominant hand index and thumb, palpate just above pubic symphysis to locate the presenting part and determine if it is engaged on the pelvis. If the presenting part is movable, it is not yet engaged. If it is not movable, it is engaged.
- Forth, while facing the maternal legs from the abdomen palpate, enter the presenting part with both hands moving towards the birth canal while applying deep pressure. When the head is the presenting part, you will feel a round prominence in one of your hands. If this cephalic prominence is on the same side as the back and spine, the fetus is in face presentation. If the prominence is on the same side as the small parts, the fetus is on vertex presentation.

## Fetal Monitoring

In the situation of imminent delivery, there is little use for advanced fetal monitoring in the ED. Nevertheless, an initial assessment of the fetal well-being is appropriate if time allows. The most basic way to assess the fetal wellbeing is by listening to the fetal heart rate (FHR). This can be done by auscultating with a stethoscope, Doppler US or bedside ultrasound, placed on the mother's abdomen and in the area where the fetal thorax is located.

The normal fetal heart rate is 110-160 BPM and should be measured over 2 minutes, as it is normally variable. Higher rates represent fetal distress. Decelerations on FHR can be normal or abnormal.

Decelerations occurring during the uterine contractions are called early decelerations and are due to the vagal response to the compression of the fetal head on the mother's pelvis.

Decelerations occurring towards the end of the contractions and peaking after the

contraction are called late decelerations and constitute a sign of fetal distress or placental insufficiency.

Decelerations occurring at any moment without relation to the contractions are called variable decelerations and represent an indication of umbilical cord compression or umbilical cord prolapse.

In the case of late or variable decelerations, the patient should be given oxygen, IV fluid bolus, placed on lateral decubitus and immediate OB consultation should be obtained as immediate emergency delivery may be indicated.

## Vaginal Exam

The effacement, dilatation, station, and position should be determined. On vaginal exam, while using lubricated sterile gloves, locate the cervix and the presenting part. Palpate the cervix to determine effacement and dilatation, palpate the presenting part to locate anterior and posterior fontanel, chin or

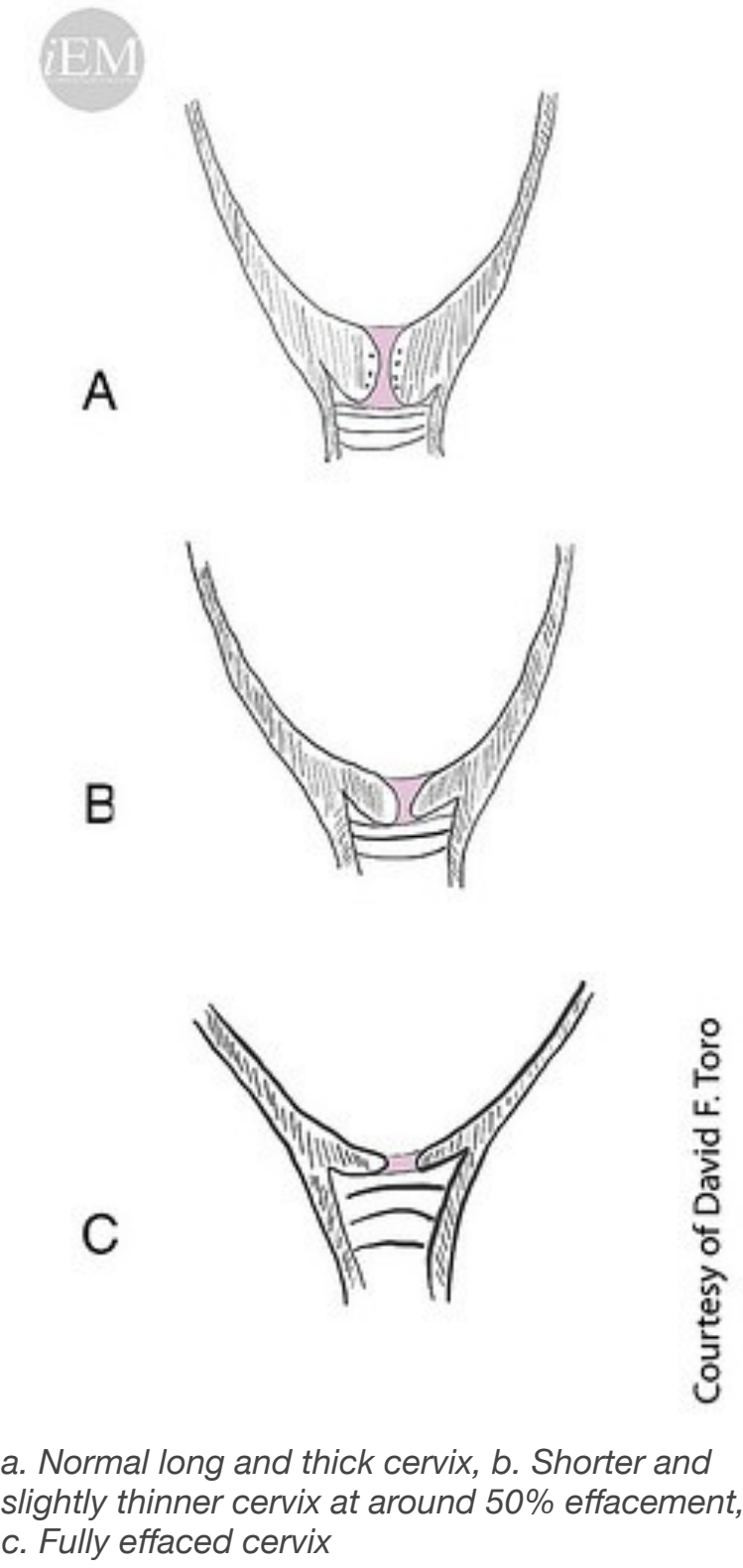
sacrum and locate the ischial spines and determine the station.

The position is the relation of the occiput or posterior fontanel in relation to the maternal pelvis. If the fetus is presenting breech, the sacrum is used as fetal reference, and if it is presenting face, the chin is the point of reference. The most common presentation and what is considered normal is left/right occiput anterior.

Effacement is the progressive thinning and shortening of the cervix that occurs slowly during early labor and progressively faster during active labor. It may occur simultaneously with dilatation, especially on multiparous women. It is measured qualitatively from 0% (long and rubbery) to 100% (very thin and soft) by palpation of the cervix. (Illustration 16.3 2)

Cervical dilatation is the measurement of the cervical os diameter, expressed in centimeters. 10cm is considered full dilatation. A cervix permeable to 1 finger is considered dilated to 1cm and if it's

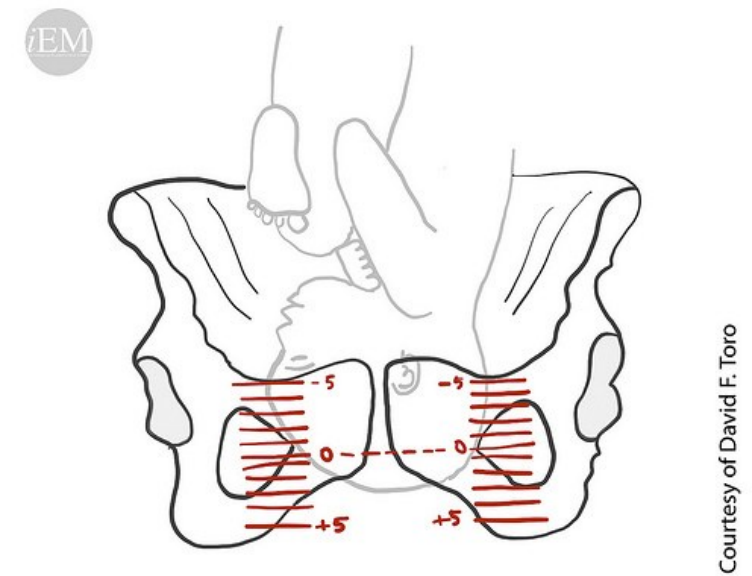
**Illustration 16.3** Effacement stages



permeable to 2 fingers is considered dilated to 3cm, which is considered active labor.

Station is the level of the presenting part in relation to the ischial spines. This measurement is done by palpation, where the ischial spines are at 8- and 4-o'clock on the vaginal canal. It is also described in centimeters where 0 is at the level of the ischial spines, negative numbers (-1, -2, -3, -4, -5) are above and positive numbers (+1, +2, +3, +4, +5) are below the spines. (Illustration 16.4).

**Illustration 16.4** Stations

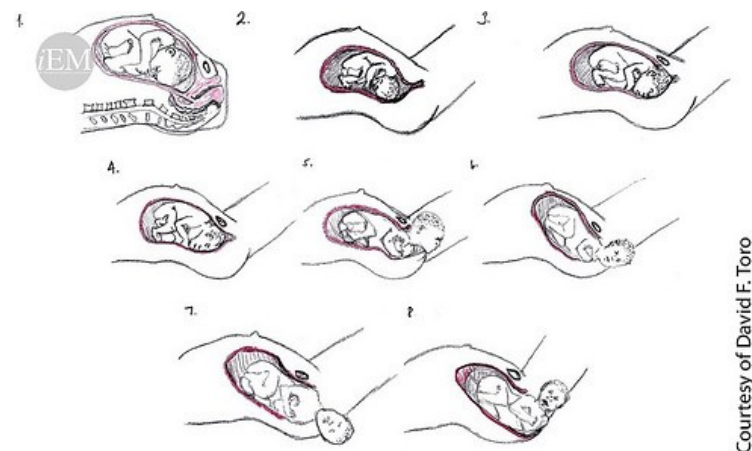




## Fetal Movements During Labor

As the fetus descends on the birth canal, several movements occur as a mechanical process where the fetus follows the path of least resistance, adapting the position of the presenting part to the dimensions of the birth canal and producing the following movements: (Illustration 16.5)

### Illustration 16.5 Fetal movements



*Fetal positions for delivery. 1. Cephalic fetal presentation before labor, 2. Engagement, 3. Flexion, 4. Internal Rotation, 5. Extension, 6. External rotation, 7. Expulsion of anterior shoulder, 8. Expulsion of posterior shoulder.*

**Engagement:** During this stage, the biparietal diameter passes through the pelvic inlet and is considered engaged when the head reaches station 0. On primigravid patient, this movement occurs in the last 2 weeks of pregnancy, but on multiparous patients, it may occur when labor begins.

**Flexion:** During this stage, the fetus neck is flexed to present a shorter diameter on the pelvis.

**Internal Rotation:** This occurs as the presenting part crosses the ischial spines. At this point, the relative transverse position on the head moves back to the original occiput anterior position.

**Extension:** The occiput reaches the vaginal introitus and passes under the symphysis pubis. During this stage, the head is born from the occipital area, the bregma, forehead, nose and finally chin, at the perineal area of the vaginal introitus.

**External rotation:** The head returns to an anatomic position in relation to the rest of the fetal torso. The head returns now to a transverse position, just as during engagement, while the fetal shoulders are passing between the ischial spines.

**Expulsion:** During this stage, the rest of the fetal body is born. The shoulders continue descending on an oblique position as they finalize their descent on the pelvis and are delivered – first the anterior shoulder and then the posterior one at the level of the perineum. The fetal pelvis is the smallest of the large fetal diameters and descends on the maternal pelvis following the same path and is delivered all at once, in contrast to the fetal head and the shoulders.

## The Delivery Procedure

The following materials are typically used for a normal vaginal delivery procedure (does not include equipment for neonatal resuscitation):

- 0-0 absorbable (Chromic catgut or undyed Vicryl<sup>®</sup>) suture material
- Basins
- Kelly clamps
- Light source
- Long needle driver
- Mask with face shield
- Material scissors
- Povidone-Iodine solution
- Shoe covers
- Sterile drapes and towels
- Sterile gauze
- Sterile gloves
- Sterile lubricant gel
- Sterile waterproof gown
- Suction device or bulb syringe
- Syringes (10-20mL), and needles (22-24 gauge)

- Tissue Forceps
- Tissue scissors
- Umbilical cord clamp

Before you begin the procedure, as there is a high risk of exposure to body fluids, remember to wear sterile gloves, mask with eye protection, waterproof sterile gown, and shoe covers.

Apply iodine solution to the perineal area and clean with sterile water, then apply sterile drapes to the patient's thighs and abdomen. It is likely that stool is expelled during the birthing process; so, additional sterile drapes should be available to prevent fecal contamination of the baby or the perineal area.

The ideal position of the patient is on a birthing table with stirrups and lithotomy position. If this is not available, additional personnel may help the patient maintain the knees flexed and the hips abducted. If a regular stretcher is used, it is helpful to place folded sheets or an inverted bedpan to elevate the patient's pelvis and

provide additional space for the delivery maneuvers.

The following videos describe and illustrate a step by step guide to the normal vaginal delivery procedure.

This [video](#) demonstrates the hand technique for spontaneous vaginal delivery on a simulated environment.

This [video](#) shows the baby's process on a virtual simulation.

Once the fetus has been delivered, carefully place him/her on the sterile drape on the mother's abdomen and stimulate while drying with sterile towels or gauze.

After drying and stimulating, clamp the umbilical cord about 1 in or 3 cm from the newborn's abdominal skin using an umbilical clamp, Kelly clamp with rubber ligature or fabric ligature. Then place a Kelly clamp about 1 in from the umbilical clamp and use the scissors to cut the umbilical cord between the two clamps.

Obtain a blood sample from the placental end of the cord for neonatal testing. At this moment, follow the neonatal resuscitation guidelines, calculate the initial APGAR scale and wrap the newborn to prevent hypothermia. Then, place the newborn under radiated heat.

## Immediate Post-Delivery Care

Delivery of the placenta occurs up to 30 to 40 minutes after fetal delivery, and it is, for the most part, a passive process. Once you see a slight increase in vaginal bleeding and the remaining umbilical cord protrudes slightly, ask the mother to bear down and apply very gentle traction on the umbilical cord to gently advance the placenta through the vaginal canal, while applying cephalad suprapubic massage to the contracted uterus to prevent uterine inversion.

Never force the expulsion of the placenta or apply more than gentle traction as umbilical cord separation and uterine inversion can cause major bleeding.

Once the placenta is delivered, inspect it to ensure it was delivered completely, and there are no remaining parts in the uterus.

After delivering the placenta, inspect the cervix, vaginal mucosa and the perineum for tears that may need to be repaired.

The main mechanism for hemostasis after the placenta has detached is uterine muscle contraction over the blood vessels, so an infusion of oxytocin, ergonovine or methylergonovine may be given to aid in the process. Oxytocin (Pitocin) is the most commonly used agent. Add 20 units to a 1 Liter Normal Saline bag and infuse at 10 mL/min until bleeding is controlled. Once bleeding is controlled, finish the infusion at 1-2 mL/min.

## Key Additional Points

A controlled and gentle delivery of every fetal part is preferred to an explosive delivery and decreases, to some extent, the probability of vaginal tears.

During delivery of the head, gentle upward pressure with a sterile towel or drape to prevent anal contamination on the perineal area helps elevate the presenting part and decrease the pressure the fetal chin exerts on the perineal skin.

Immediately following delivery of the head, palpate the fetal neck to inspect for umbilical cord encircling the neck. This cord needs to be reduced over the fetal head before delivery can continue.

As with any other procedure, don't stand too close to the patient as fluids may be suddenly expelled risking contamination.

Be very careful when holding the newborn, as he/she will be very slippery. It is advisable to hold him/her close to your body.

**References and Further Reading**, click [here](#)

# Pericardiocentesis

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by David Wald and Lindsay Davis

## Case Presentation

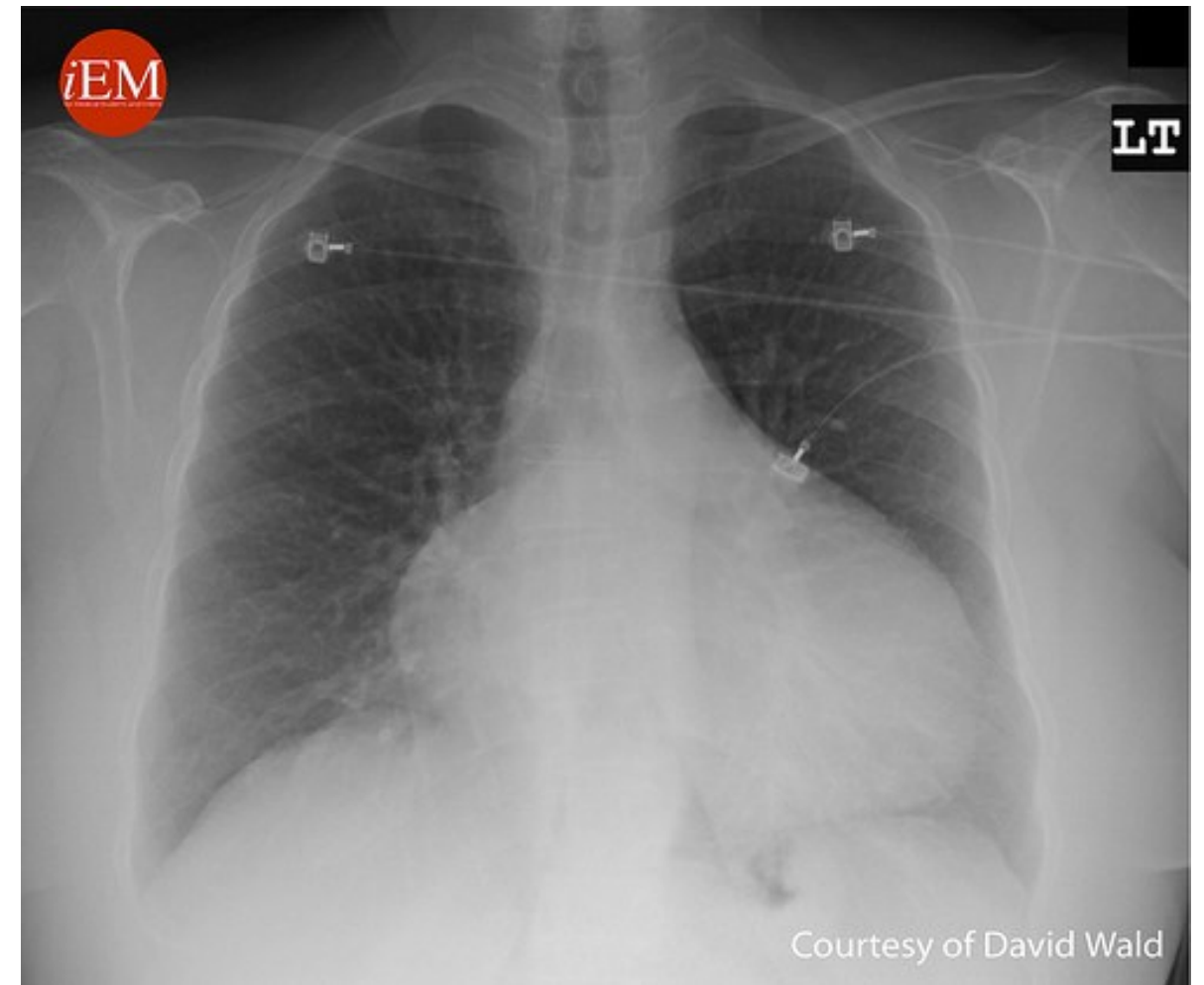
*A 52-year-old female with a history of metastatic breast cancer presents to the emergency department with a complaint of progressive shortness of breath throughout the past week. She initially felt tired and out of breath with walking and climbing stairs. This has progressed to the point where she would feel short of breath just walking around her house. Over the last day, she noted that it has become*

*difficult lying flat in bed. Today she is experiencing shortness of breath at rest. She reports no other active health conditions. Her initial vital signs are blood pressure 92/66 mmHg, heart rate of 108, respiratory rate of 20 per minute, temperature 37.1°C and a room air oxygen saturation of 96%. On evaluation, the patient appears uncomfortable and mildly dyspneic. She has JVD at 5cm. Her heart sounds are regular without a murmur but*



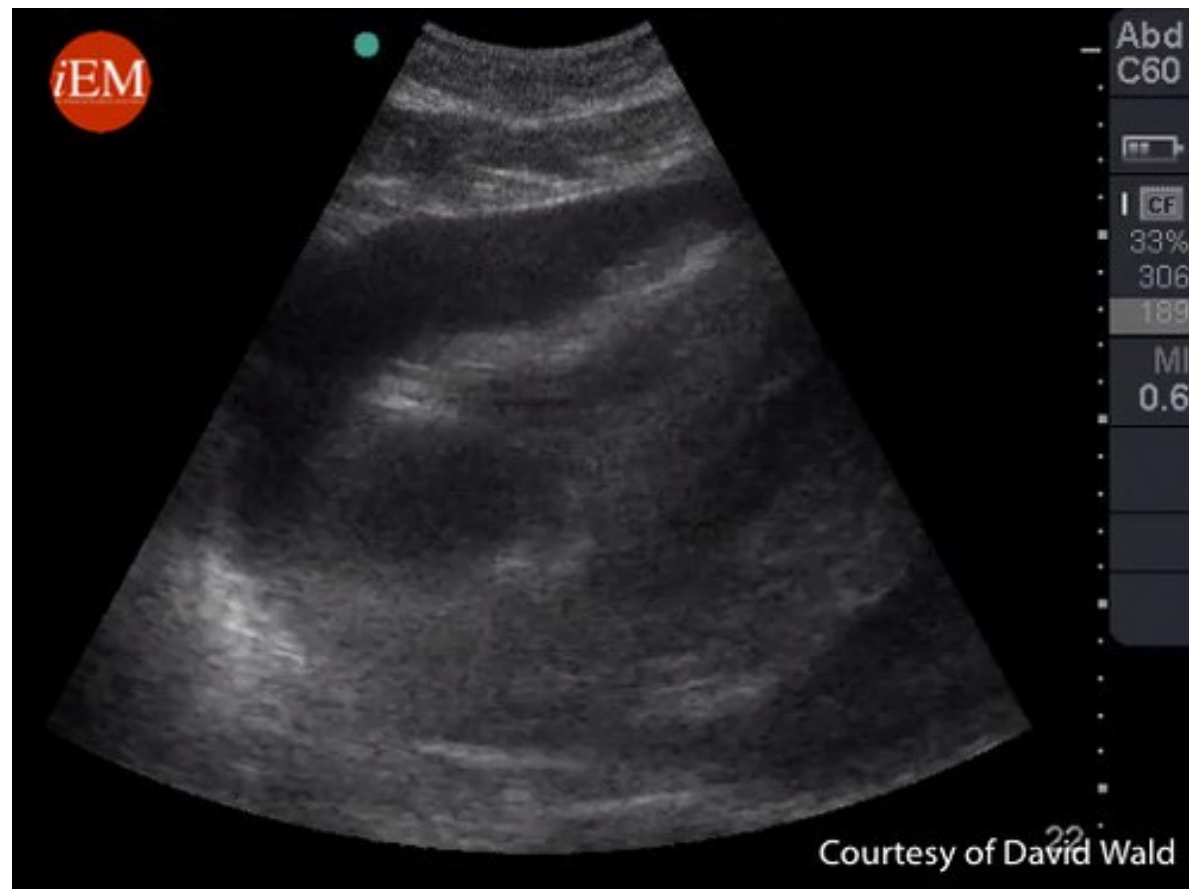
are barely audible. Lungs are clear in all fields. Her abdomen is soft and non-tender. She has no lower extremity edema. She is awake, alert and moves all of her extremities equally. A 12 lead electrocardiogram shows sinus tachycardia with low voltage in the limb leads. Her chest radiograph is shown in Image 16.7. An emergency medicine bedside cardiac ultrasound is performed, see Image 16.8.

Image 16.7



Pericardial effusion on chest radiograph demonstrating an enlarged “water bottle” shaped heart. This finding can be seen in pericardial effusions that have developed slowly, allowing time for the pericardial sac to stretch.

**Image 16.8**



*Echocardiography of a pericardial effusion in the subcostal 4-chamber view. Note the inward bowing of the right ventricle indicating cardiac tamponade and hemodynamic compromise.*

## Pathophysiology and Indications

The heart is surrounded by a double layer fibrous sac known as the pericardial sac. The first layer, the visceral pericardium, is adherent to the cardiac epicardium. The second layer, the parietal pericardium, is separated by the visceral pericardium by 25-50 mL of physiologic serous fluid, allowing the heart to beat without friction.

A pericardial effusion develops when fluid accumulates in the potential space between the visceral and parietal pericardium. Pericardial effusion can be caused by a number of conditions including trauma, malignancy, uremia, cardiac rupture, and infectious causes such as tuberculosis and viral pathology. The clinical effect of pericardial effusion can vary based on etiology, volume, and particularly the speed at which the effusion accumulates. If fluid accumulates very gradually, the pericardium can remodel and stretch to accommodate the increased volume. In these cases, symptoms are often insidious and progressive over days to weeks. Alternatively, if fluid accumulates suddenly, as in the case of penetrating chest trauma, the pericardium is not able to stretch to accommodate the increased volume of fluid. The result can be the rapid development of pericardial tamponade and death.

Pericardial tamponade occurs when the pressure of a pericardial effusion becomes greater than the pressure in the right atrium, resulting in the collapse of the right atrium during diastole. Remember, the right side of the heart is a low flow system, and it does not take much pressure to impede flow. The increased pressure within the pericardial space can eventually cause compression of the entire right side of the heart, leading to the restricted ventricular filling. This, in turn, can lead to decreased stroke volume and decreased cardiac output. If not treated, this can result in hypotension, cardiogenic shock, and death. If an effusion develops rapidly, as little as 150 mL of fluid, can cause

tamponade physiology. A pericardial effusion that restricts cardiac output resulting in tamponade is a true cardiovascular emergency and is the primary indication for emergency pericardiocentesis.

At times, making the diagnosis of pericardial tamponade can be difficult because the condition can present in an insidious fashion if the effusion accumulates slowly over many days to weeks. In these cases, the differential diagnosis will include many other causes of shortness of breath. Alternatively, pericardial tamponade should always be considered as a potentially reversible cause in the patient who develops cardiac arrest shortly after chest trauma.

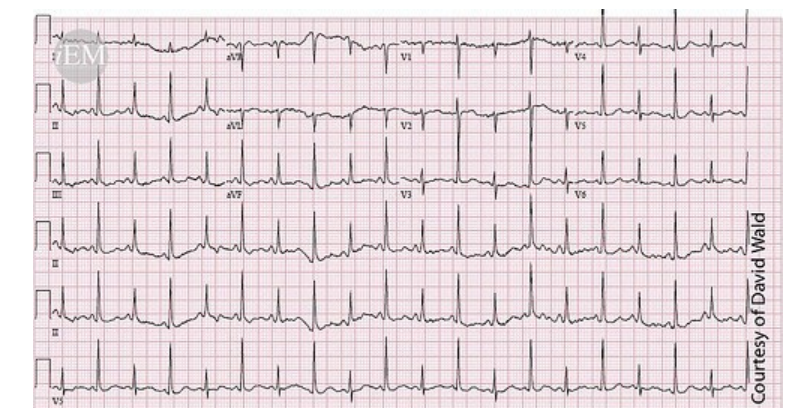
Cardiothoracic surgeon Claude Schaeffer Beck originally described the physical exam findings of pericardial tamponade in 1935 as what is now commonly known as Beck's triad: muffled heart sounds, jugular venous distension (JVD), and hypotension. Though these findings are

described in every textbook discussion of pericardial tamponade, studies have shown that these exam findings have poor sensitivity and specificity and are present together with a minority of the time. The most common signs and symptoms exhibited by patients with tamponade include dyspnea, tachycardia, JVD, and a narrowed pulse pressure. Pulsus paradoxus (a decrease in systolic blood pressure of greater than 12 mmHg during inspiration) has been found to be one of the more sensitive and specific exam findings associated with cardiac tamponade. However, the tedious and time-consuming nature of this exam technique makes it difficult at best and often an impractical tool for diagnosing a life-threatening condition in an unstable patient in the emergency department.

Electrocardiographic findings can include low voltage QRS complexes, PR segment depression, ST elevation and electrical alternans (see image 16.9), though these findings are also not specific or sensitive. Chest radiographs are equally

nonspecific. In the case of a chronic pericardial effusion, a chest radiograph may demonstrate an enlarged "water bottle" shaped cardiac silhouette (see image 16.7). However, if the effusion is from an acute traumatic etiology, the pericardial sac will not have had time to stretch to accommodate the increased volume, leaving the cardiac silhouette unchanged.

### Image 16.9



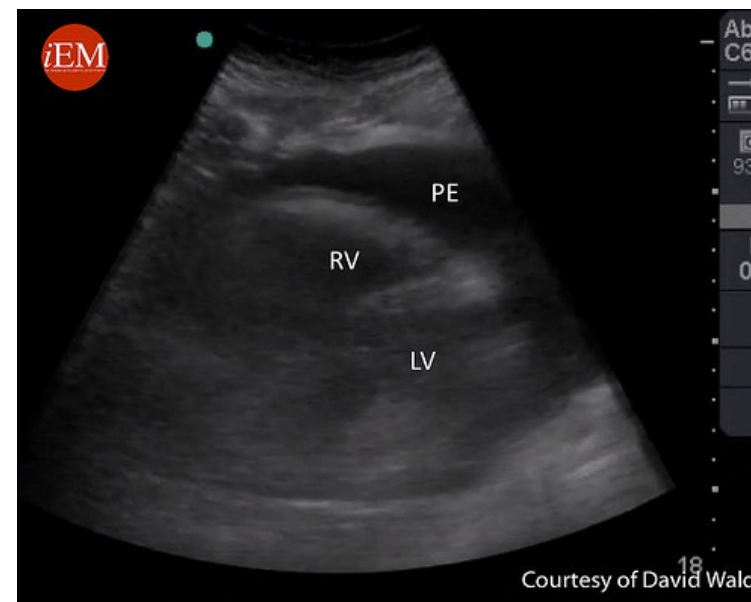
*Electrocardiogram demonstrating electrical alternans in a patient with a large pericardial effusion. Note the alternating QRS amplitude due to the swinging motion of the heart within the pericardial sac.*

Ultrasound is the best and most applicable diagnostic imaging modality used to identify a pericardial effusion or



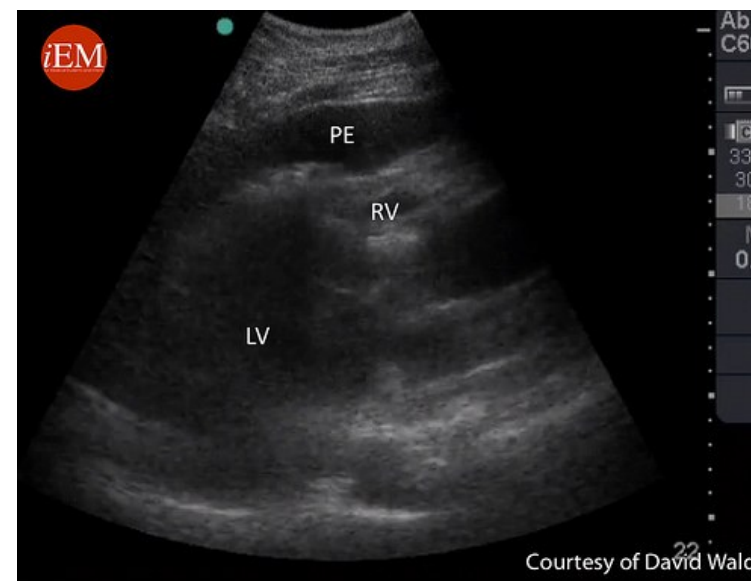
tamponade. It is noninvasive and safe, with no risk of radiation to the patient. The increased availability of bedside ultrasound in the emergency department has allowed for instant point of care diagnosis of this potentially life-threatening condition. In addition to offering direct visualization of the effusion itself, sonography allows the operator to assess for hemodynamic compromise secondary to increased pericardial pressure. The initial sign of this process is the collapse of the right atrium in diastole, followed by bowing of the right ventricle. A pericardial effusion on ultrasound appears as a dark (anechoic) stripe between the myocardium and the pericardium. Pericardial effusion can typically best be viewed by one of three commonly used cardiac windows: 1) subxiphoid, 2) parasternal long axis, and 3) apical 4-chamber. Ultrasound images of a pericardial effusion from these three views are seen in Image 16.10-11.

**Image 16.10**



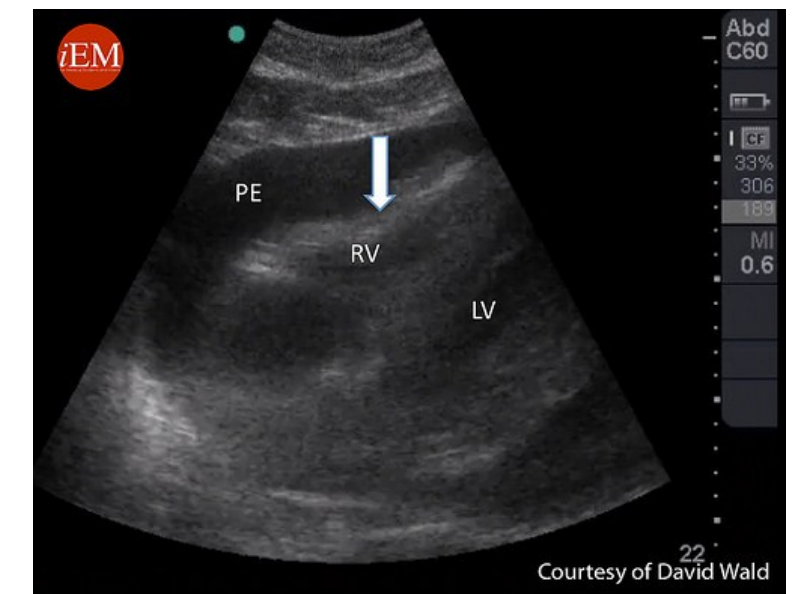
*Echocardiography of a pericardial effusion (PE) in the subxiphoid view. Note that the effusion is seen as a large anechoic stripe surrounding the heart.*

**Image 16.11**



*Echocardiography of a pericardial effusion (PE) with tamponade physiology as demonstrated by the collapse of the right ventricle (RV) seen in parasternal long-axis view.*

**Image 16.12**



*Echocardiography of a pericardial effusion (PE) in the apical 4-chamber view. The arrow indicates collapse of the right ventricle (RV) seen in cardiac tamponade.*

## Contraindications

### Absolute contraindication

Aortic dissection.

### Relative contraindications

- Uncorrected coagulopathy or anticoagulant therapy
- Thrombocytopenia (platelets <50)
- Traumatic hemopericardium (managed surgically)



## Equipment and Patient Preparation

If the time permits and the patient is awake, the procedure should be explained to the patient, and the physician performing the procedure should obtain written informed consent.

Cardiopulmonary resuscitation equipment should be readily available in the event of a life-threatening arrhythmia or further hemodynamic decompensation.

Intravenous sedation should be considered but must be reconciled with the urgency of the procedure and the patient's hemodynamic stability.

If possible, elevate the chest wall 30-45%; this brings the heart itself closer to the chest wall.

All providers involved in the procedure should wear sterile protective clothing including gown, gloves, and mask.

## Equipment

- Sterile gowns, gloves, masks, and caps for all providers in the room
- Sterile drapes
- Sterile ultrasound transducer cover
- Sterile ultrasound gel
- Chlorhexidine sponge
- 1% lidocaine with epinephrine
- 25 gauge needle (1.5 inches) and 10mL syringe for lidocaine injection
- 16 or 18 gauge spinal needle (5-10cm)
- 1 empty 5mL syringe
- 5 mL syringe filled with 8cc of saline
- 3-way stopcock
- 60 mL syringe
- Central venous access kit/  
Pericardiocentesis kit
- Sterile gauze

## Procedure Steps

Before bedside ultrasound was readily available, practitioners would perform emergent pericardiocentesis in a “blind” fashion, relying on anatomic landmarks to guide the placement and direction of the needle. This approach can put surrounding structures at greater risk of injury. Bedside ultrasound allows direct visualization of the heart, not only leading to faster, more accurate diagnosis of tamponade, but it also allows practitioners to choose the approach that offers them the best access to the pericardial effusion while avoiding surrounding structures. The three most commonly used ultrasound approaches are the Subxiphoid, parasternal and apical approach (see Images 16.13-16.15). Details regarding probe positions and the pros and cons of each approach can be found in Table 1.

Image 16.13



*To achieve a subxiphoid view of the heart, place the probe inferior to the xiphoid process and angle it cephalad and towards the patient's left.*

Image 16.14



*For a parasternal long approach, position the probe to the left of the sternum, in the 4th or 5th intercostal space.*



*To use an apical approach, place the probe slightly lateral to the midclavicular line, in the 5th or 6th intercostal space.*

Table 16.1 shows. probe positions for ultrasound-guided pericardiocentesis

**Table 16.1** Probe positions for ultrasound guided-pericardiocentesis

APPROACH	PROBE POSITION	PROS	CONS
Subxiphoid	Inferior to the xiphoid process, angling the probe cephalad and left towards the heart	Good acoustic window created by liver, no overlying bony structures	Risk of hepatic puncture
Parasternal	To the left of the sternum, in the 4th or 5th intercostal space	No overlying organs	Risk of damage to internal mammary artery, which lies 3-5 cm lateral to sternal border
Apical	Slightly lateral to midclavicular line, in 5th or 6th intercostal space. Insert needle above the rib to avoid neurovascular bundle and aim toward right shoulder	The part of the heart closest to the needle is the left ventricle, which is thick walled and will suffer less significant injury if inadvertently penetrated. The coronary arteries are also smallest at the apex.	Often most challenging view due to body habitus, increased risk of pneumothorax

1. Clean and drape the patient in a sterile fashion.
2. Prepare the ultrasound transducer with a sterile sheath.
  - The ideal probe for this procedure is the 5 to 1 MHz transducer due to its small footprint.



Please watch [video 1](#) and [video 2](#).

## Hints and Pitfalls

- Do not confuse the epicardial fat pad with a pericardial effusion. Keep in mind that significant effusions are circumferential, and the fat pad is only an anterior structure. Also, fat pads will move with each contraction, whereas a pericardial effusion does not and thus will appear to change size as the ventricular wall constricts inward away from the pericardium with each contraction.
- Use the ultrasound to measure the depth of the pericardial effusion, and make sure you use a needle that is long enough.
- Keep your needle tip in view at all times! This may require tilting or adjusting the probe to maintain visualization around surrounding structures as you progress.
- Rapid drainage of pericardial effusion can lead to rapid increases in preload,

- If that is not available, the 5 to 2 MHz curvilinear probe should provide adequate views.

3. Visualize the area of maximal pericardial effusion with the ultrasound probe.
4. If time allows and the patient is awake, infiltrate the area of planned needle insertion with 5 mL of 1% lidocaine.
5. Using an in-plane approach, insert the needle at a 45-degree angle (for subxiphoid approach 30-45 degrees to prevent liver puncture), maintaining negative pressure on the syringe while keeping the needle tip in view on the screen at all times.
6. Observe the needle entering the pericardial sac.
7. Once the pericardial sac has been penetrated, inject agitated saline and look for the bubbles visible on ultrasound in the pericardial sac to confirm correct placement.

8. Once placement is confirmed, proceed with Seldinger technique

- remove the syringe from the needle,
- insert the guidewire into the needle tip and thread the wire.
- Once the guidewire is in place, remove the needle, keep the wire in place.
- Thread a dilator over the guidewire.
- Remove the dilator, keep the wire in place.
- Slide a catheter over the wire. When the catheter is in place, the guidewire can be removed.

9. Aspirate/drain the pericardial effusion/ blood.

10. Check the vitals and check with ultrasound.



which can rarely cause flash pulmonary edema, bradycardia and rebound hypertension.

- If there is a question about the source of bloody aspirate, look for clotting ability. If the blood is from a traumatic effusion or intracardiac, it will clot easily. If the blood has migrated into the pericardial space and results from a non-traumatic effusion, it will be defibrinated and thus will not clot or will take much longer to clot.
- Agitated saline can be prepared by connecting two 5mL syringes to the needle catheter via a 3-way stopcock valve. One syringe contains saline, the other air. Agitate the saline by rapidly pushing the saline from one syringe to the other with the stopcock closed to the needle catheter. The saline is sufficiently agitated when it appears cloudy.

## Post Procedure Care and Recommendations

- Immediately after the procedure, a chest x-ray should be obtained to ensure there is no pneumothorax or air under the diaphragm.
- If a pigtail catheter was inserted for continuous drainage, it should be sutured in place. Avoid tying the sutures so tightly that it occludes the catheter.
- Cover the catheter insertion site with sterile gauze and tape.
- The patient should remain on a cardiac monitor.
- Vital signs and cardiac rhythm should frequently be reassessed to monitor for findings that would suggest re-accumulation of the effusion and inadvertent procedural complications.

## Complications

Most complications of pericardiocentesis are related to needle penetration of either the heart or surrounding structures. Studies have cited serious complication rates of 20-30% using the blind approach. The use of ultrasound has

significantly decreased these complications. A study of 1127 ultrasound-guided pericardiocentesis at Mayo Clinic showed a procedure success rate of 97%. The rate of major complications, which were those requiring intervention, was 1.2%. The minor complication rate was 3.5%. Similar findings have been repeated in more recent studies.

## Specific complications to be aware of include:

- Dry tap (often caused by blockage of the needle with clot or tissue)
- Dysrhythmias, though the literature suggests dysrhythmias related directly to the pericardiocentesis procedure itself are rare
- Myocardial or coronary artery puncture leading to hemopericardium
- Liver laceration
- Pneumothorax/hemothorax
- Pneumopericardium

- Vascular injury, most likely the internal mammary artery and the intercostal neurovascular bundle
- Flash pulmonary edema from rapidly increase in left ventricular preload after pericardial decompression
- Suppurative pericarditis
- Costochondritis

**References and Further Reading**, click [here](#)

# Lumbar Puncture

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by Khuloud Alqaran

## Case Presentation

*A 16-year-old male, without a known case of any medical illness, presented to the ED accompanied by his mother. His chief complaint was altered mental status. Three days earlier to his presentation, he had a fever, nausea, vomiting, and headache. The symptoms worsened over time. His mother noted that 2 weeks earlier he visited his grandmother at the intensive care unit. On physical examination, he opened his eyes once the doctor called his name; then, he said, “Where am I, what is the time?” He was moving in the bed with no neurological focal deficit. Vital signs as following: Temperature 38C, heart rate of 110/min and blood pressure of 100/45 mmHg. Nuchal rigidity was positive, and he had skin rashes over his shins as shown in the picture below.*

**Image 16.16**



Courtesy of Dr. DB Calheiros. Retrieved from <http://www.atlasdermatologico.com.br/disease.jsf?diseaseId=287>

## Indications

### Diagnostic

- Presumption of meningitis
- Presumption of subarachnoid hemorrhage (SAH).
- Presumption of any syndrome such as Multiple sclerosis or Guillain-Barre
- Presumption of Pseudotumor Cerebri (Idiopathic Intracranial Hypertension,

IIH) by measuring the opening pressure of the Cerebrospinal fluid (CSF)

### Therapeutic

- Administration of anesthetic spinal agent or intrathecal drug (e.g., Chemotherapy in case of Leukemia).
- Drain away excess of CSF in case of IIH

## Contraindications

### Absolute contraindication

Infected skin or soft tissue at the entry site of the needle

### Relative contraindication

- Increased Intracranial Pressure (ICP)
- Deranged coagulation profile (INR > 1.4 or Severe thrombocytopenia, platelet < 40,000)

### Other contraindication

Brain abscess or any space-occupying lesions (SOL)

Lumbar puncture should not be delayed for any reason in the clear indication. However, in some cases, further

investigations may be needed with the CT scan.

### Indications for CT head prior to the LP are as follows;

- Age > 60 years
- Immunocompromise status
- Altered mental status or presence of any neurological deficit
- Any sign/symptoms of elevated ICP (e.g., headache, papilledema, or bradycardia)
- History of the Central nervous system (CNS) lesion (e.g., old stroke, SOL such as tumor or abscess)
- Recent seizure activity (within a week from presentation)

## Equipment and Patient Preparation

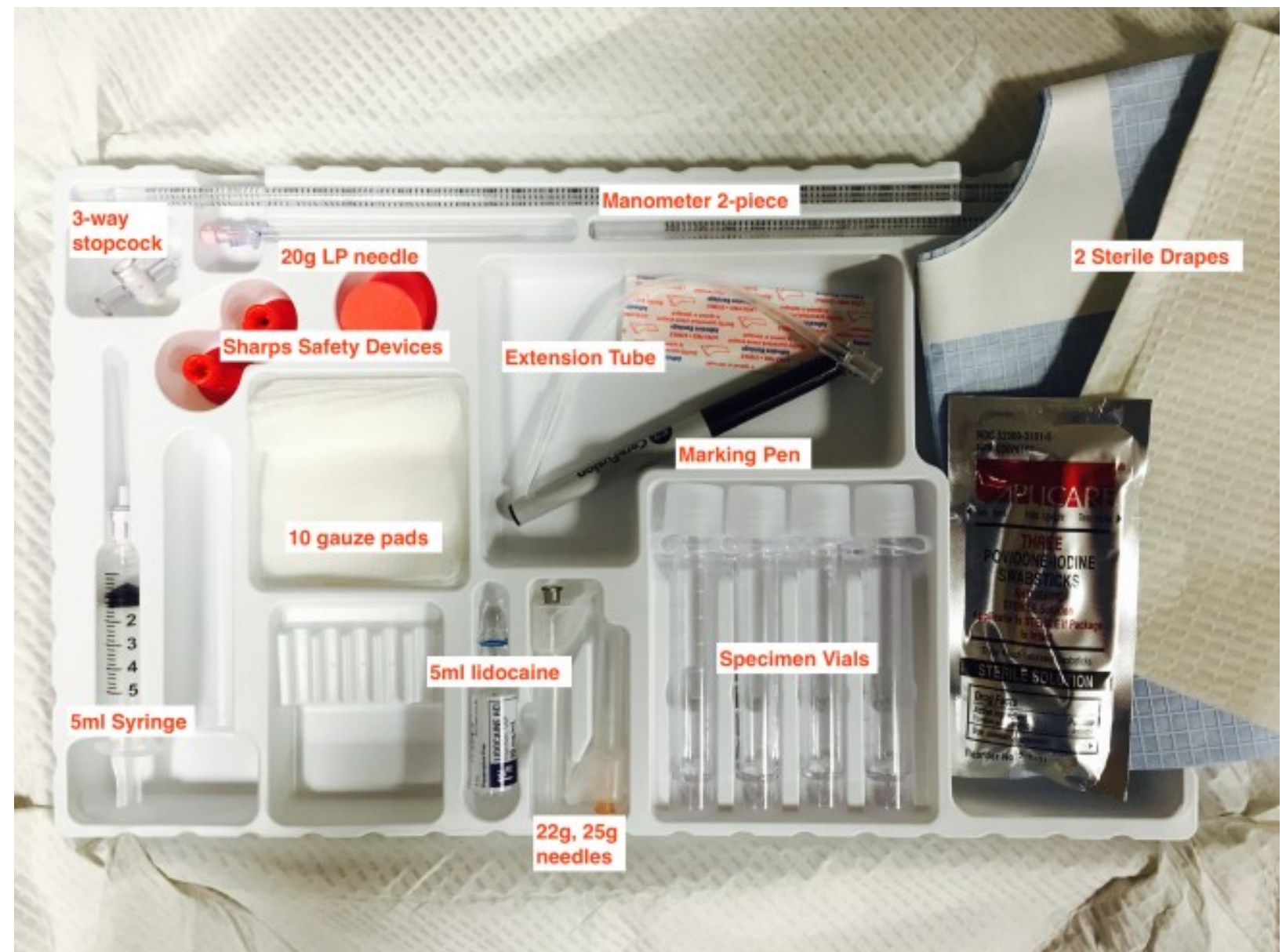
### Equipment



Pre-packed LP procedure kit may include the following items:

- Sterile dressing
- Sterile drape
- Alcohol Swabs
- Spinal needle size 18 gauges
- Lidocaine 1% without epinephrine
- Syringe 3mL
- 3 Way stopcock
- Monometer
- 4 plastic test tubes (numbered from 1-4)
- Syringe 10mL
- 2x2 Gauze
- Small Adhesive plaster
- Extra supplies
  - Non-sterile marking pen
  - Chlorhexidine swabs for sterilization

**Image 16.17** LP set



Retrieved from <https://i2.wp.com/blogs.brown.edu/emergency-medicine-residency/files/2015/08/Figure-2.jpg>

Maintain universal precautions (sterile gloves, surgical facemask, cap and sterile gown)

The spinal needle should be replaced by Stylet spinal needle size 20 or 22 gauge. However, the length should be chosen depending on the patient's age.

- Infant 1.5in or 3.8cm
- Child 2.5in or 6.3cm
- Adult 3.5in or 8.8cm

The registered nurse or any doctor colleague to help position the patient during the procedure

Place/order CSF tests needed prior to the procedure

## Patient preparation

- Informed consent needs to be taken from the patient or the legal guardian.
- Speak to your patient during the procedure; the patient is already anxious and can't see what you are doing. Talk to the patient and explain what you are doing in a calm manner. For example, say: " Now I am going to numb the site with a smaller needle; it may have a burning sensation. Please don't move."
- Patient placed in two positions upon preference and patient condition or age

- Lateral recumbent with spine parallel to bed: in this position, the patient's hips, knees, and chin are flexed toward his/her chest (fetal position). Analgesia, sedation or anxiolytic (e.g., benzodiazepine) can be considered if appropriate to reduce patient anxiety.
- Sitting upright with hips flexed with feet on a stool: in this position, the patient is awake and cooperative. It's preferred in obese patients when it would ease midline localization. The patient would sit upright; his/her lumbar spine should be perpendicular to the table. His/her foot should be supported by a stool and not hanging down. A pillow can be placed at the patient lap so he/she can bend forward and keep his/her chin towards his/her chest (angry cat position).

## Procedure Steps

1. Position the patient as mentioned above
2. Identify the landmarks anatomically by palpating the midline vertebral

columns of L4 at the level of the posterior superior iliac crests. (Adult injection site – any interspinous space from L2-S1 as the spinal cord terminates at L1 level. However, pediatrics injection site should be only from L3-L4/L4-L5 interspinous space as the conus medularis ends at the level of L1-L3).

3. Use a skin-marking pen to approximate the entry site.
4. Gown up and maintain universal precautions (sterile gloves, surgical facemask, and head cap).
5. Apply the antiseptic solution in a circular motion starting from the entry site to the periphery.
6. Apply the sterile drape.
7. Create a skin wheal of 1% Lidocaine; make sure it is no more than 1mL to avoid losing the landmark. Then inject into the deeper tissues.

8. Advanced the needle at the midline with your dominant hand holding the hub and your non-dominant hand supporting the needle by placing the thumb/index finger on the shaft of the needle for balance, parallel to the bed. The angle should be facing upward, aiming at the umbilicus.
9. Characteristic “pop” is occasionally felt when the needle passes the dura. If there is no sound, draw the stylet periodically checking for the CSF after approximately 4-5 cm.
10. Once CSF starts to drain, attach the manometer to measure the pressure. Then, start collecting the fluid from tube number 1 to 4 in sequence pattern. No more than 1mL in 1-3 tubes, then 3-4mL in tube 4.
11. Replace the stylet before removing the needle; then, remove both of them together.
12. Cover the injection site with the adhesive plaster.

Please watch [video 1](#) and [video 2](#).

## Hints and Pitfalls

- Like any other procedure, preparation is a must. Position the patient, palpate his back, get to know his anatomy, then mark it with a marking pen.
- Your patient is elderly, and you are hitting a bone only after insertion of 25% of your needle. In most patients, the needle should be inserted 50-75% of its length prior to obtaining CSF flow.
  - You may be hitting calcified supraspinal ligament.
  - Try to enter from the lateral aspect to avoid the calcified ligament.
- The patient can get very anxious and alarmed.
  - Talk to the patient; he can't see what you are doing. Tell him what step you are going to do.

- Ask someone to help you hold the patient and maintain his position during the procedure.
- If still anxious, give him/her some anxiolytic or even sedation if necessary.
- Injecting lidocaine can sometimes obscure your landmark.
  - Try not to inject more than 1mL to make a wheal; then, inject the remaining in the deeper tissues.
- The traditional teaching “feel the first pop then the second pop, CSF will flow.”
  - Never depend on the pop. Most of the time a series of pops are felt instead, as several spinal ligaments are encountered prior to entering the space.
- In obese patients,

- Use the full length of the needle to 3.5-inch, or use the 6-inch “harpoon” needle.
- Try to do it in a sitting position.
- CSF is red or tinged red
- The needle is too deep and hits a venous plexus leading to a traumatic tap. On the other hand, it’s subarachnoid hemorrhage or meningitis.
- Signs of a traumatic tap
  - The absence of xanthochromia (shows up within 12 hours and persists 2-4 weeks). If present at the time of the tapping then its highly suggest SAH.
  - RBC count 400-500 RBCs or less suggestive of the traumatic tap. Must become zero at one of the last tubes.

- RBC counts taper down from tube 1 to tube 4. This is not fully reliable unless it is completely clear by the 4th tube, but classically, the RBC count decreases by 30%.
- Examining the 4th tube as a separate entity can also help rule out SAH
- <100 RBC: almost certainly traumatic
- <500 RBC: probably traumatic
- >10K RBC: likely SAH
- Is it bacterial or viral?
  - There are couple measures helping us to identify the cause. These are
    - Opening pressure (cmH2O)
    - WBC count (per mm3)
    - Neutrophils (%)
    - Glucose (mg/dL)

- Protein (mg/dL)
- Culture or gram stain
- Normal values include 5-20 cmH2O opening pressure, equal to or less than 5 WBC per mm3, no neutrophils, 50-80 mg/dL glucose and 20-45 mg/dL protein.
- The bacterial CSF sample shows elevated opening pressure >500 WBC per mm3, >80% PMNL, low glucose (<40 mg/dL), and increased protein (>50 mg/dL). In addition, culture or gram stain indicates bacteria. The viral sample, however, shows normal or slightly elevated opening pressure, 100 – 500 WBC, neutrophils less than 50% and lymphocytic predominance, normal glucose, protein is normal or slightly elevated, and culture or gram stain indicates the virus.

## Post-procedure Care and Recommendations



- The patient may lie flat; however, there is controversial evidence that it may reduce headache incident.
- Vital sign should be recorded depending on the hospital guidelines.
- Neurological examination at least every 4 hours within the first 24 hours.
- Encourage fluid intakes up to 3L/24hrs (tea and caffeine may help).
- Monitor the puncture site for any bleeding, CSF leakage or infection.
- Ensure the patient void after 8 hours post procedure.
- Administer analgesia accordingly.
- Do not forget the documentation.

## Complications

Post-LP headache is the most common complication. It results from the reduction of CSF below the cisterna magna. It can't be prognostic or prevented. Starts within the first 48hrs. A mild headache, self-limited, only needs conservative therapy

(e.g. bed rest, oral analgesia, in some cases caffeine drinks can help). A refractory headache, an epidural blood patch is recommended. Using non-cutting, smaller diameter needle can decrease the occurrence. 20-22 gauge atraumatic needles are the best choice.

## Other complications are;

- Infection
- Herniation syndrome
- Formation of subarachnoid epidermal cyst
- Backache and radicular syndrome
- Spinal epidural hemorrhage

**References and Further Reading,** click [here](#)

# Nasogastric Tube Placement

---

by Sara Nikolić and Gregor Prosen

## Case Presentation

*A 47-year-old man presents to your ER complaining of nausea and vomiting. He tells you that vomiting started a couple of hours after eating dinner the night before. It was a normal vomit, consist of digested food; however, it is not associated with meals. He has barely eaten during the past 36 hours. The pain consists of cramping and is vaguely umbilical, but it is not well localized. He gets mild relief from vomiting and says the pain is severe (9/10). He has felt generally unwell and has not taken his temperature. None of his close contacts have reported any vomiting. His last bowel movement was yesterday morning, and he cannot recall passing any flatus today. About 20 years ago he had an appendicectomy.*

*On examination, the patient is lying in bed in some discomfort from the stomach cramps. He has an appendicectomy scar*

*and mild distension of his abdomen. You hear high-pitched bowel sounds on auscultation.*

*Vital signs are as follows:*

*temperature of 36.6°C, heart rate of 66 bpm, blood pressure 126/63 mmHg, respiratory rate 11/min, oxygen saturation 98% on room air.*

*Blood tests come back and show: white cell count  $7.7 \times 10^9$  cells/L, CRP 23 mg/L, Na 137 mM, K 3.7 mM, AST 27 U/L, ALT 23 U/L, ALP 29 U/L, amylase 152 U/dL, urea 4.2 mM, creatinine 92  $\mu$ M.*

*The bedside ultrasound shows distended (>3 cm diameter)*

*small bowel with numerous valvulae conniventes and increased peristalsis with whirling motion of the bowel contents.*

See [video](#).

## Introduction

Nasogastric (NG) tube placement is one of the most common procedures performed in intensive care settings, the emergency department, and hospital wards. It is frequently used for the management of patients who require decompression of the gastrointestinal (GI) tract, diagnosis and assessment, nutritional support and medication administration. The procedure is rapid, simple, and straightforward. The goal is to conduit the NG tube to the stomach through the nasal cavity, nasopharynx, oropharynx, and esophagus that enters the stomach below the diaphragm. The very vascular nasal mucosa lines the nasal cavity. This is important to remember because, after each unsuccessful insertion, incidences of mucosal bleeding and hemodynamic complication increase.

## Procedure

## Indications

### Diagnostic

- Evaluation of upper gastrointestinal (GI) bleeding
- Identification of the esophagus and stomach on a chest x-ray

### Procedural/Therapeutic

- Aspiration of gastric fluid content
- Administration of radiographic contrast to the GI tract
- Gastric decompression
- Relief of symptoms and bowel rest in the small-bowel obstruction
- Gastric content aspiration for recent toxic ingestions
- Administration of medications such as active charcoal

- Feeding
- Bowel irrigation
- NG tube can be kept following corrosive ingestion for the development of a tract in the esophagus. It subsequently can be used for balloon dilatation.

### Contraindications

Absolute contraindications for NG tube placement are:

- Severe midface trauma – rare perforation through the thin cribriform plate of the ethmoid bone and into the brain can occur. Patients with facial trauma are best served with orogastric intubation.
- Recent nasal surgery

Relative contraindications for NG placement are:

- Coagulation abnormality
- Esophageal varices (usually, a Sengstaken-Blakemore tube

introduced, but an NG tube can be used for lower-grade varices) or stricture

- Recent banding of esophageal varices
- Alkaline ingestion (the tube may be kept if the injury is not severe)

## Equipment and Patient Preparation

### Equipment

- Choose the size of nasogastric tube that is appropriate for the patient. A size 16 to 18 French is typically used for an adolescent or adult patient.
- Personal protective equipment
- A water-soluble lubricant is often enough to facilitate passage.
- If topical anesthesia is available: 1.5 mL of atomized lidocaine may be atomized into the nasopharynx, with 3 mL applied to the oropharynx and swallowed, or 5 mL of 2% lidocaine jelly can be injected



into the nostril. Do not use oil-based lubricant.

- Cup of water with a straw
- Emesis basin and towel
- Tongue blade and flashlight
- Aspirating and irrigating syringes (10ml and Toomey syringe, 60 mL)
- Tincture of benzoin, scissors, and tape or semipermeable transparent membrane dressing
- pH indicator paper
- Stethoscope
- Suction tubing and container
- Wall suction, set to low intermittent suction

### General patient preparation

- Give the patient nothing by mouth for several hours (ideally).

- Explain the procedure to the patient, including route, purpose, and anticipated duration of intubation.
- Have the patient sit upright or raise the head of the bed. If this is not possible, passing the tube with the patient in the left lateral decubitus position has less risk of aspiration than if the patient is supine.
- Check for nasal obstruction or maxillofacial trauma. Have the patient inhale briskly through each nostril and use the more patent nostril for intubation.
- Test the gag reflex. Patients unable to gag are at increased risk of pulmonary aspiration.

### Procedure steps

- Wash hands
- Introduce yourself and confirm patient details
- Explain the procedure and gain consent

- Gather equipment for the procedure
- Inspect the nostrils for septal deviation – to determine which nostril is more patent, ask the patient to occlude each nostril and breathe through the other
- Position the patient sitting upright with their neck partially flexed
- Put the gloves on
- If available, put the anesthetic spray to the back of the patient's throat for comfort
- Estimate the length of insertion by measuring the distance from the tip of the nose, around the ear, and down to 5cm below the xiphisternum. This point can be marked with a piece of tape on the tube.
- Ask the patient to hold the cup of water and put the straw in his or her mouth.
- Lubricate the tip of the NG tube
- Place the kidney plate near the patient in case there is leakage.

- Warn patient you will start the procedure and in case of pain they should tip you on the hand.
- Gently insert the NG tube along the floor of the nose. Advance NG tube parallel to the nasal floor (not angled up into the nose) until it reaches the back of the nasopharynx, where resistance will be met (10-20 cm).
- At this moment, ask the patient to sip water through the straw and start swallowing. With each sip you continue to advance the NG tube until the distance of the previously estimated length is reached.
- Confirm the tube's placement in the stomach by radiographic imaging. Alternatively, gently aspirate gastric contents with a 3-cc syringe, and check the pH. A pH < 4 suggests the tip is in a gastric location. A pH > 5 does not reliably predict location because the respiratory system and intestinal tract distal to the pylorus often have a pH > 5. Verifying tube position by

auscultating a rush of air over the stomach using the 60 mL Toomey syringe is not as helpful because the sounds of air in the bronchial tree can be mistaken for gastric insufflation.

- Apply benzoin or another skin preparation solution to the nose bridge. Tape the NG tube to the nose to secure it in place.
- If clinically indicated, attach the tube to wall suction after verification of correct placement.
- Dispose of used equipment into a clinical waste bin and wash hands.
- Explain to patient that the procedure is over. Reassure that the NG tube will become more comfortable over the next few hours. Offer patient paper towels to clean their face and nose. Document clearly the procedure.

Please watch [video 1](#) and [video 2](#).

Withdraw the nasogastric tube if, at any time resistance, respiratory distress, the

inability to speak, or significant nasal hemorrhage occurs.

## Hints and Pitfalls

- During insertion, if concern exists that the NG tube is in the wrong place, ask the patient to speak. If the patient can speak, then the tube has not passed through the vocal cords and/or lungs.
- To improve the success rate of nasogastric tube placement, provide external and medially directed pressure on the ipsilateral neck at the level of the thyrohyoid membrane. It will collapse the piriform sinus and eliminate it as a potential site for impaction. This maneuver was successful for difficult nasogastric intubation in 85 percent of patients.
- The nasogastric tube may coil in the oropharynx, mouth, or hypopharynx. Cool the tube in cold tap water or ice water for 5 minutes to make the tube stiffer and then reinsert it. A larger bore tube may be inserted more easily. A final option is to place several fingers

through the patient's mouth and into the oropharynx. The fingers can be used to guide the tube against the posterior oropharyngeal wall and into the hypopharynx. Do not attempt this unless the patient is unconscious or paralyzed to prevent them from biting the fingers.

- The risk for tube misplacement is greater in the intubated patient who is unable to assist with nasogastric intubation. Observe that there are no changes in the patient's oxygen saturation when inserting the nasogastric tube. It is very easy for the nasogastric tube to pass by the cuff of an endotracheal tube without much resistance.
- GlideScope facilitates NG tube insertion and reduces the duration of the procedure in anesthetized patients. Also, esophageal guidewire-assisted insertion with manual forward laryngeal displacement technique most frequently results correct positioning of the NG

tube in anesthetized and tracheally intubated patients after the first attempt.

- American Association of Critical-Care Nurses partook a survey about feeding tube practices in adult intensive care units. The recommendations were to obtain radiographic confirmation that each blindly inserted tube is correctly positioned before the first use, which is currently not adequately implemented. Also, auscultation is widely used despite recommendations to the contrary.

## Post Procedure Care and Recommendations

- The patient should be able to speak without respiratory distress immediately after placement of the nasogastric tube. Observe the patient for complaints of neck pain, substernal chest pain, dysphagia, drooling, trismus, fever, or subcutaneous and mediastinal air. These would be signs of esophageal

perforation or errant placement of the nasogastric tube.

- Although auscultation of air in the stomach has been classically used to determine correct placement, air insufflated into the pleural space or the esophagus after misplacement of the tube can be just as easily heard over the upper abdomen.
- Gastric contents should be able to be aspirated through the nasogastric tube.
- Testing the pH of the gastric contents can help predict the placement of the nasogastric tube. However, in one trial, pH of 4 was able to accurately identify the location of only 56% of all NG feeding tubes when compared with the reference standard radiography. The use of H2 blockers makes the assessment of gastric pH difficult. Radiographic demonstration of the tube in the antral or fundal portion of the stomach is the preferred method of confirmation.

## Complications

- The most common complication of nasogastric intubation is discomfort in the nasopharynx and oropharynx.
- Placement in the nares can result in epistaxis if the nasal mucosa is irritated, abraded, or ulcerated.
- These complications can be reduced or avoided with generous lubrication of the nasogastric tube and the installation of topical anesthetics and vasoconstrictors.
- Sinusitis may occur from the nasogastric tube obstructing the sinus ostia. These complications are usually of no clinical significance.
- A more serious consequence of nasogastric intubation is misplacement into the respiratory tree. This is estimated to occur in up to 15% of cases. The incidence increases in frequency with a patient who has a diminished gag reflex or a decreased level of consciousness. The presence of

a cuffed endotracheal tube does not preclude passage into the respiratory tree. The nasogastric tube will pass the cuff of the endotracheal tube without significant resistance. Advancing the tube into the airway can result in perforation of a bronchus or the lung and result in a pneumothorax, hydropneumothorax, pulmonary hemorrhage, empyema, or bronchopulmonary fistula. These complications are increased if medication or alimentation is infused into the respiratory tree.

- The most serious complication of nasogastric tube placement is the esophageal perforation. This most often occurs in the posterior wall of the cervical portion of the esophagus and through the cricopharyngeus muscle. Risk factors for esophageal perforation include a preexisting esophageal abnormality, altered mental status, cervical osteophytes, cardiomegaly, tracheal intubation, a rigid nasogastric tube, and multiple attempts. Perforation

often results in mediastinitis with a subsequent mortality rate of up to 30%. Prompt recognition, surgical repair, and parenteral antibiotics can reduce the mortality rate to less than 10%. The use of softer and smaller nasogastric tubes with generous lubrication can reduce the risk of esophageal perforation.

## Pediatric, Geriatric, Pregnant Patient, and Other Considerations

The placement of a nasogastric tube in children is often difficult. Their large tonsils and adenoids may hinder the passage of the nasogastric tube. These tissues are soft, easily injured, and may bleed as the nasogastric tube is passed. The tongue, large by comparison with adults, may push into the oropharynx and impede the passage of the nasogastric tube. Their nostrils and nasal passage are quite small and limit the size of nasogastric tube that may be passed. Also, size is calculated by the formula  $((\text{age in years} + 16) / 2)$ . Typical sizes include 8 French for infants, 10 to 12



French for small children, and 12 to 14 French for older children. Most common complications are nasal ala pressure sores that are usually not associated with significant morbidity and mortality.

**References and Further Reading**, click [here](#)

# Procedural Sedation and Analgesia

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by Nik Rahman

## Introduction

When working in the emergency room, one often finds himself in a situation where painful diagnostic or therapeutic procedures are needed to be performed. These procedures cause major pain and anxiety to the patient and using local anesthesia on its own does not suffice in some situations. Procedural sedation reduces anxiety, pain and potential unpleasant memories associated with such procedures while also facilitating the execution of the procedure. Therefore, procedural sedation and analgesia (PSA) has become a fundamental and required skill for emergency physicians to learn for day to day use. This chapter is also important for many physicians who are facing painful procedures.

## Definition

Procedural sedation is defined as the use of short-acting analgesic and sedative agents in order to enable clinicians to perform procedures effectively while monitoring the patient closely for potential adverse effects.

## Terminology

Anxiolysis is a state in which decreasing apprehension regarding a particular situation without affecting patient's level of awareness.

Without the intentional alteration of mental status, relief of pain is called Analgesia. However, the altered mental state may be a secondary effect of the medications administered for this purpose.

Dissociation is a trancelike cataleptic state induced by an agent such as Ketamine and is characterized by profound analgesia and amnesia. In this state, protective reflexes, spontaneous respirations, and cardiopulmonary stability are preserved.

The controlled reduction of environmental awareness is called sedation.

## Levels of Sedation

1. Minimal sedation (anxiolysis) refers a patient in this state responds normally to verbal commands, although cognitive functions and coordination may be impaired. Respiratory and cardiovascular functions are unaffected as this state essentially involves mild anxiolysis or pain control.
2. Moderate sedation/analgesia involves depression of consciousness while patients still respond purposefully to verbal commands or light tactile stimulation. Airway, ventilation and cardiovascular functions are all spontaneously maintained. The patient

may appear somnolent but is arousable to voice or light touch. (Reflex withdrawal from the painful stimulus is not considered a purposeful response.)

3. Dissociative sedation is a cataleptic state induced by a dissociative agent (i.e., Ketamine). This state is characterized by profound analgesia and amnesia. This state is achieved while airway protective reflexes are maintained along with spontaneous respiration. Cardiopulmonary stability is also maintained.
4. Deep sedation/analgesia is a state where the patient has a depressed level of consciousness in which he/she requires painful or repeated stimulation to evoke a purposeful response. Patients may require assistance to maintain a patent airway. Spontaneous ventilation may be inefficient. Cardiovascular function is usually preserved.

5. General anesthesia refers to the drug-induced loss of consciousness which patients are not arousable to painful stimulation. In this state, the ventilatory function is often impaired, and assistance may be required to maintain the airway and respiration. Positive-pressure ventilation may be required as spontaneous ventilation is often impaired. Cardiovascular function may be impaired as well.

The different levels of sedation need to be understood as each scenario may require a certain level to be achieved to facilitate the performance of the required procedure.

## Steps for PSA

Early planning and preparation are key to preventing adverse events that may occur and can be catastrophic if procedural sedation is poorly managed.

Adequate staffing needs to be ensured; this is done by having a nurse or another qualified individual present for continuous monitoring of vital signs and airway

patency as well as having a separate provider performing the procedure.

### Pre-procedural evaluation

The patient should be evaluated for sedation. PSA may not be suitable for every patient in the ED. This can be done objectively by using the ASA classification and difficult airway assessment (Table 16.2).

**Table 16.2** American Society of Anesthesiologists Physical Status Classification

CLASS	DESCRIPTION	EXAMPLES	SEDATION RISK
I	Normal and healthy patien	No past medical history	Minimal
II	Mild systemic disease without functional limitations	Mild asthma, controlled diabetes	Low
III	Severe systemic disease with functional limitations	Pneumonia, poorly controlled seizure disorder	Intermediate
IV	Severe systemic disease that is a constant threat to life	Advanced cardiac disease, renal failure, sepsis	High
V	Moribund patient who may not survive without procedure	Septic shock, severe trauma	Extremely high

Anesthesiology consultation may be required for a patient with an anticipated difficult airway or an ASA classification of III. It may be wise to have the anesthesiologist perform the sedation in the operating room which is a better-controlled environment.

### Consent

When possible, a written consent should be obtained that discusses the risks, benefits, and potential side effects of PSA. The patient or direct family members need to sign the consent before the procedure takes place.

### Equipment Preparation

1. High-flow oxygen source: ASA guidelines recommend considering oxygen for moderate sedation and strongly recommend it for deep sedation.
2. Suction should be prepared in case any secretions accumulate in the airway, which in turn need to be suctioned.
3. Airway management equipment: there may be a need for airway support during PSA. An appropriate size endotracheal tube should be prepared with an intubating blade, an oral airway and a bag valve mask (BVM).
4. Monitoring equipment includes a pulse oximeter, ECG monitor/defibrillator, transcutaneous pacing pads, Blood pressure monitor, and Capnography. Capnography measures end-tidal carbon dioxide (CO2) partial pressure. The ASA recommends



the use of capnography for monitoring patients during PSA whether they are on supplemental oxygen or not. The aim is to detect if the patient's ventilatory drive is affected during the procedure in order to perform corrective measures when needed.

#### 5. Vascular access equipment

## Medications used in PSA

### ETOMIDATE

Etomidate is a fast-acting sedative with little analgesic effect. The onset of action is usually within 1 minute with a short duration of action. It lasts between 3 to 5 minutes with standard dosing. Elimination is done rapidly by the liver; therefore, duration of action may be longer in patients with liver failure. It has few hemodynamic effects, and its neutral cardiovascular profile makes it one of the most appealing agents for use.

The contraindication is hypersensitivity to the medication.

Etomidate is a Pregnancy Category C medication

Side Effects:

- Muscle twitching is a well-known side effect that is generally well tolerated
- Nausea and vomiting may occur after emergence

- Respiratory depression and hypoxia are possible

Generally, Etomidate has been shown to be safe and effective when used for procedural sedation.

### KETAMINE

Ketamine is a rapidly acting dissociative anesthetic that also produces a profound analgesic effect. Doses can be repeated and titrated to effect with no risk of cumulative adverse events. Onset, duration, and dosing vary according to the route of administration.

Contraindications:

- Hypersensitivity to the medication
- Can lead to a hyper-sympathetic state, which might be deleterious especially in patients with Coronary Artery Disease
- Avoided in patients who are predisposed to psychotic behavior

Ketamine is a Pregnancy Category C medication

Side Effects:

- Ketamine is a derivative of the street drug Phencyclidine; it causes an increase in systemic and pulmonary blood pressures, heart rate, cardiac output, cardiac workload and myocardial oxygen demand. It should be avoided in elderly or patients with cardiac diseases.

- Most common side effect seen with Ketamine is the emergence phenomenon. It occurs in approximately 15% of patients and is mild in almost all of them. Less than 1 to 2% of patients have significant emergence agitation.
- Transient airway laryngospasm (0.4%)
- Emesis

## FENTANYL

A rapid-acting synthetic opioid administered intravenously. Duration of action may last from 30 to 60 minutes. It is a pure analgesic with no sedative properties; therefore, it must not be used alone for PSA.

It is a Pregnancy Category C medication

Side Effects:

- Respiratory depression is more likely at higher doses
- Hypotension and bradycardia are rare but may occur with high doses

## PROPOFOL

This is an ultra-short-acting sedative-hypnotic agent that has no analgesic properties. It is quickly cleared from the body, permitting superior titration, earlier recovery, and discharge. It also possesses potent antiemetic properties and decreases intracranial pressure. Because of lack of analgesic effect, it

should be preceded by an opioid when performing painful procedures.

Contraindications: in patients with allergy to eggs or soy

Side Effects:

- Respiratory depression
- Apnea
- Hypotension
- Pain over the injection site

## MIDAZOLAM

Midazolam is a benzodiazepine sedative, amnestic and anxiolytic agent with no analgesic properties. It is usually combined with opioids like Fentanyl to provide a good combination of sedation and analgesia during PSA. It is eliminated by hepatic metabolism and renal excretion; therefore, prolonged effects may be seen with dysfunction of any of those two organs.

Side Effects:

- Cardiopulmonary depression

It is a Pregnancy Category D medication

The following table 16.3 summarized the list of all medications used during procedural sedation.

**Table 16.3** Agents Used in Procedural Sedation

AGENT	RECOMMENDED DOSE	ONSET OF ACTION	DURATION OF ACTION	SIDE EFFECTS AND COMMENTS
Etomidate	In adults 0.2 mg/kg over 30 to 60 seconds	< 1 minute	3 to 5 minutes	Advantages: no cardiovascular or respiratory depression Disadvantages: action is too short for some procedures
Ketamine	1 to 2 mg/kg intravenously 4 to 5 mg/kg intramuscularly	1 minute 5 minutes	10 to 20 minutes 15 to 45 minutes	Nystagmus, hypersecretions, agitation, emergence delirium, vomiting, myoclonus, laryngospasm, cardiovascular stimulation Contraindications: hypertension, ischemia, psychosis, infants younger than three months
Fentanyl	Initial dose in adults: 1 to 1.5 mcg/kg intravenously Titrate: 1 mcg/kg every 3 minutes intravenously	1 to 2 minutes	30 minutes	Cough, hiccup, itching, vomiting, respiratory depression Requires another agent for sedation, repeat dosing may be required
Midazolam	Initial dose in adults: 0.02 mg/kg Titrate: 1 mg intravenously every 3 minutes Initial dose in children (6 months - 5 years of age): 0.1 mg/kg	1 to 2 minutes	30 minutes	Respiratory depression, hypotension Requires another agent for analgesia, poor reliability, repeat dosing may be required
Propofol	1 mg/kg Intravenously, then 0.5 mg every 3 minutes if needed	15 to 30 seconds	5 to 10 min	Respiratory depression, apnea, hypotension, pain over injection site Advantages: Rapid onset, short duration, antiemetic, cerebral protective. Contraindications: allergy to eggs or soy

## Reversal Agents

Fentanyl and Midazolam both have antagonists that can be used to reverse their effects.

Agents like Naloxone for opioids (i.e., Fentanyl) and Flumazenil for benzodiazepines (i.e., Midazolam) are the ones commonly used for this purpose. However, the routine use of reversal agents should be avoided as the duration of the sedation agents may

exceed that of the reversal agents. If used, it should be followed by an extended observation period to ensure recovery.

Caution: when using Flumazenil, it may lead to Status Epilepticus, especially in patients with unidentified benzodiazepine use or in patients with a known seizure disorder.

## Recovery and Discharge

Finally, it is important to monitor all patients until the moment of recovery. Drowsy patients should not be left unattended. Patients should be monitored until they spontaneously wake up and are able to perform their normal functions independently. Complete recovery to baseline function may not be necessary for discharge. Generally, an awake patient who is able to drink without vomiting, able to ambulate and voids normally is capable of going home ideally with family members or friends as an escort. Appropriate discharge instructions should be given.

**References and Further Reading**, click [here](#)



# Rapid Sequence Intubation (RSI)

by Qais Abuagla

## Introduction

Airway management is one of the most important skills for an Emergency Department practitioner to master because failure to secure an airway can lead to mortality or morbidity. Therefore, all junior doctors and trainees should be aware of this procedure in the early of their career.

RSI is an advanced airway management technique that induces immediate unresponsiveness (induction agent) and muscular relaxation (neuromuscular blocking agent). It is the method of choice for intubations in the Emergency Department due to its high success rates and the fewest complications.

## Indications for RSI

- Failure of airway maintenance or protection or anticipated deteriorated clinical course
- Failure of ventilation or oxygenation
- Minimize oxygen consumption and optimize oxygen delivery (e.g., sepsis)
- Prevent secondary brain injury or terminate seizure (status epilepticus)

## Contraindication

### Absolute

- Total upper airway obstruction
- Total loss of facial/oropharyngeal landmarks

### Relative

- Anticipating difficult airway

- Cardiac/respiratory arrest (this will go to crash intubation)

## Steps of RSI (7 Ps)

1. Preparation & Plan
2. Preoxygenation
3. Pre-treatment
4. Paralysis and induction
5. Protection and positioning
6. Placement with proof
7. Post-intubation management

### Preparation

- Equipment (tube, blade, Oxygen, suction, capnography, monitoring (ECG, BP, SpO<sub>2</sub>))
  - Peds tube size: (age+4) /4 or use Braselow tape
  - The depth of the tube: size x 3
- Asses for difficult airway, and set plan B for failed airway

- Assessment of airway: Anticipating difficulty in establishing an airway in emergency patients is the first step in avoiding major complications. This helps us to think about alternatives of RSI. For example, neuromuscular paralysis should generally be avoided in patients with a high level of intubation difficulty.

### LEMON

**L – Look externally:** Look for external markers of difficult intubation; these may include the following body habitus, head and neck anatomy (short neck), mouth (small opening, loose teeth or prominent teeth), jaw abnormalities (significant malocclusion), and beards.

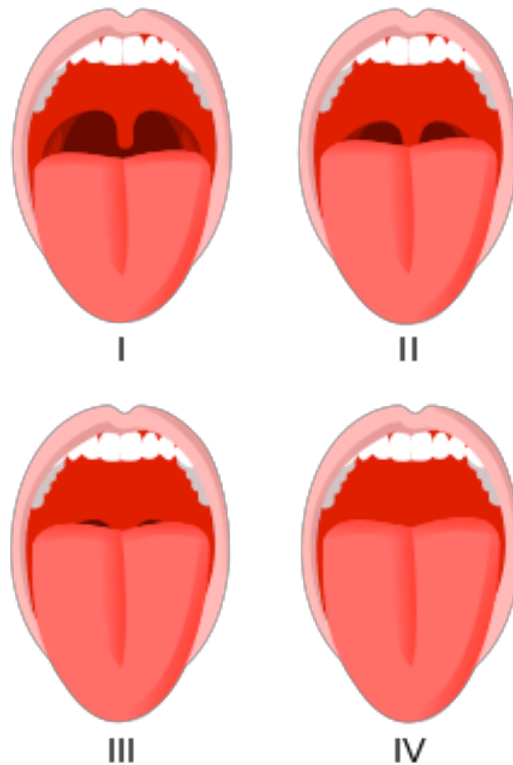
**E – Evaluate 3-3-2:** 3-3-2 rule is to assess the patient's airway geometry to determine his or her suitability for direct laryngoscopy. Can the patient fit 3 fingers between the incisors? For optimum glottis visualization, an adequate mouth opening is required. Is the mandible length 3 fingers from the mentum to the

hyoid bone? Submandibular space is adequate to accommodate the tongue making the visualization of the glottis easy. Is the larynx low enough in the neck to be accessible? The distance from the hyoid to the thyroid. 2 fingers are what we are looking for.

**M – Mallampati:** Oral access is assessed with the Mallampati scale. Visibility of the oral pharynx ranges from complete visualization, including the tonsillar pillars (class I), to no visualization at all, with the tongue pressed against the hard palate (class IV). Class I and class II predict adequate oral access, class III predicts moderate difficulty, and class IV predicts a high degree of difficulty. (illustration 16.6)

**O – Obstruction or obesity.** Upper airway obstruction can make visualization of the glottis, or intubation itself, mechanically impossible. This may present as stridor, inability to swallow secretions or alteration in voice quality. Conditions such as epiglottitis, head and neck

## Illustration 16.6 Mallampati



Retrieved from <https://commons.wikimedia.org/wiki/File:Mallampati.svg>

cancer, Ludwig's angina, neck hematoma, foreign body or thermal injury can compromise laryngoscopy, the passage of the endotracheal tube (ETT), BMV, or all three.

N – The NECK mobility. Neck mobility is desirable for any intubation technique and is essential for positioning the patient for optimal direct laryngoscopy. Neck

extension is the most important maneuver, and simple extension may be as effective as the “sniffing” position in achieving an optimal laryngeal view. Neck mobility can be significantly reduced in patients with trauma (cervical collar) or the elderly and those with arthritis.

Equipment and Preparation [video part 1](#) and [video part 2](#).

### Preoxygenation

Three minutes on 100% nonrebreather mask or six vital capacity breaths provides 8 minutes of adequate oxygenation during apnea, but this time period decreases in pregnancy, obesity, and extreme of ages.

Maintaining SpO<sub>2</sub> > 90% differs between patients.

- Healthy 70 kg adult 8 minutes
- Moderately ill adult 5 minutes
- 10 kg child 4 minutes
- Obese adult 3 minutes

• Very ill patient <2 minutes

### Pretreatment

It is used to blunt the adverse effect of laryngoscopy and intubation but scant evidence

### Medication of pretreatment

#### Atropine

- 0.02 mg/kg IV
- For all children <10 to prevent bradycardia

#### Fentanyl

- 2-3 mcg/kg IV
- For elevated ICP, myocardial ischemia, aortic dissection, subarachnoid hemorrhage

#### Lidocaine

- 1.5mg/kg
- For increased ICP and bronchospasm

## Indication for pretreatment: **PREMED**

- Pediatric
- Resistance (asthma)
- Elevated ICP
- MI
- Elevated BP
- Dissection

## Paralysis and Induction

You don't want an awake paralyzed patient!!

First induction agent is given, then it is followed by a paralytic agent. The induction agents main aim is to induce rapid loss of consciousness to facilitate ease of intubation.

## Medication for induction

### Etomidate

- 0.2-0.3 mg/kg IV

- Rapid action, and short duration. It has no analgesic effect.
- It's the most hemodynamically stable induction agent. This is an advantage over other agents in shock, anaphylaxis or any case where the further drop in blood pressure can be catastrophic.
- Etomidate has a potential cerebroprotective effect as it decreases cerebral metabolic oxygen consumption and reduces cerebral blood flow and intracranial hypertension while maintaining cerebral perfusion pressure
- Side effects are nausea, vomiting, myoclonus and adrenal cortical depression with multiple doses.

### Ketamine

- 1-2mg/kg IV, 4-5mg/kg IM.
- NMDA receptor antagonist.
- Ketamine produces a loss of awareness within 30 seconds, peaks in

approximately 1 minute, and has a clinical duration of 10 to 15 minutes.

- Many protective reflexes are preserved with Ketamine, including airway reflexes. Ketamine has a direct bronchodilator effect and causes catecholamine release. Therefore, it is mainly used in patients with asthma, anaphylaxis and hemodynamically unstable patients. Because of its features, it's an excellent alternative to etomidate.
- Side effects are the raise of BP (avoid in elderly) and emergence phenomena (visual, auditory, proprioceptive and confessional illusions which may progress to delirium after waking up from sedation)

### Propofol

- 1.5 to 2.0 mg/kg IV,
- It produces significant venous dilation, myocardial depression and can reduce cerebral perfusion pressure.



- Because of the propensity of propofol to cause hypotension, through both vasodilation and direct myocardial depression, the dosage is reduced, or the drug is avoided altogether in hemodynamically compromised patients.

**Other agents:** Benzodiazepines like midazolam and barbiturates like thiopental and methohexital

### Paralytic agents

Neuromuscular blocking agent (NMB) are mainly divided into depolarizing agents (DPA) and non-depolarizing agents (NDPA)

### Succinylcholine

- 1.5 -2 mg/kg.
- It is the only DPA used in the emergency room having a rapid onset and short half-life. It takes 45-60 seconds to induce paralysis and takes 8-10 min to recover.

- It can rise serum potassium levels, and is contraindicated in the following circumstances:

- Hyperkalemia
- Patient  $\geq 5$  hours post burn
- Patient  $\geq 5$  days post crush injury or denervation
- Neuromuscular diseases (amyotrophic lateral sclerosis, multiple sclerosis, muscular dystrophy)
- Denervation (stroke, spinal cord injury)  $> 5$  days until 6 months post injury
- Intra-abdominal sepsis  $> 5$  days until resolution

### Rocuronium

- 1-1.2 mg/kg.
- NDPA.
- It has a comparable time to paralysis but a longer recovery time of 35-45

minutes. It can be reversed by Sugammadex which can be an advantage in some circumstances.

A [video](#) about RSI drugs.

### Protection and Positioning

**In-line Stabilization:** In cases of trauma in which cervical spine injury is suspected and not yet ruled out, protection of the cervical spine is a priority and intubation must be performed without movement of the head. An assistant is required to maintain inline stabilization. This allows the cervical collar to be opened giving better access. The head and neck are maintained in the neutral position.

If no cervical spine injury is suspected flexing the neck and extending the head to the so-called sniffing position helps to align the axes and facilitates visualization of the glottic opening.

### Placement with proof

Intubation should be performed carefully and gently. After flaccidity is achieved laryngoscopy glottis is visualized, the

clinician places the endotracheal tube between the cords, inflates the cuff, withdraws the stylet, and confirms placement.

A [video](#) about intubation details.

Intubation [video](#).

Confirmation of proper endotracheal tube (ETT) placement is crucial; unrecognized esophageal intubation leads to devastating complications.

### **Confirm the placement by a combination of**

- Visualizing the passage of the ET tube between the cords
- Listening to both sides of the chest and over the stomach
- End-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) determination (either colorimetric or quantitative).

### **Post-intubation management**

After intubating the patient, the tube is tied or taped in place. Maintaining sedation is essential; infusions should be

prepared and started as soon as possible. A post-procedural chest x-ray is obtained to confirm the depth of tube placement and to evaluate for evidence of barotrauma as a consequence of positive pressure ventilation. Oro-Gastric tube and urinary catheter insertion used to decompress the stomach and monitor urine output respectively.

**References and Further Reading**, click [here](#)

# Reduction of Common Fractures and Dislocations

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by Dejvid Ahmetović and Gregor Prosen

## Introduction

Most of the orthopedic injuries can be predicted considering the chief complaint, the age of the patient and the mechanism of the injury itself. Additionally, a careful physical examination and the patient's history can often predict radiographic findings with great accuracy. If an injury is suspected by clinical examination but cannot be completely confirmed by evaluating the radiograph, the patient should be treated as if the injury is present and discharged with detailed instructions on how to look out for any additional signs of neurovascular complications, compressions, and cast care.

Injuries to the musculoskeletal apparatus include one or more of the following structures: bone, joints, ligaments, tendons and in some cases vasculature and nerves.

Simple definitions of the injuries to the musculoskeletal system include the following:

**Fracture:** A disruption of bone tissue, which may be caused by the application of a force that exceeds the strength of the bone tissue itself, repetitive stress to the bone tissue, or invasive processes that weakens the bone structure and integrity.

Dislocation: Complete disruption of a joint, whereby articular surfaces are forced from their normal position, which immobilizes the joint temporarily. In the case of a subluxation, there is still a partial contact of the articular surfaces.

## Fractures

Orthopedic injuries commonly result from accidents and often involve otherwise healthy individuals, especially in the younger population. Accurate diagnosis and treatment are of great importance both economically and medically.

Most fractures result from excessive force applied to otherwise healthy bone tissue, resulting in disruption of the bone cortex. Disruption may occur from a variety of forces, including a direct blow, axial loading, bending forces, torque forces and combinations of these.

All fractures are either simple or multifragmentary (communited). A simple fracture (spiral, oblique or transverse), is a single circumferential disruption of any part of the diaphysis, metaphysis or articular surface. A multifragmentary fracture (communited) is any fracture with one or more completely separated fragments, which can be further classified as either wedge or complex. A wedge fracture consists of fragments that, after reduction, main fragments still have some contact between themselves; in a complex fracture, however, one or more intermediate fragments and main fragments are no longer in contact after reduction.

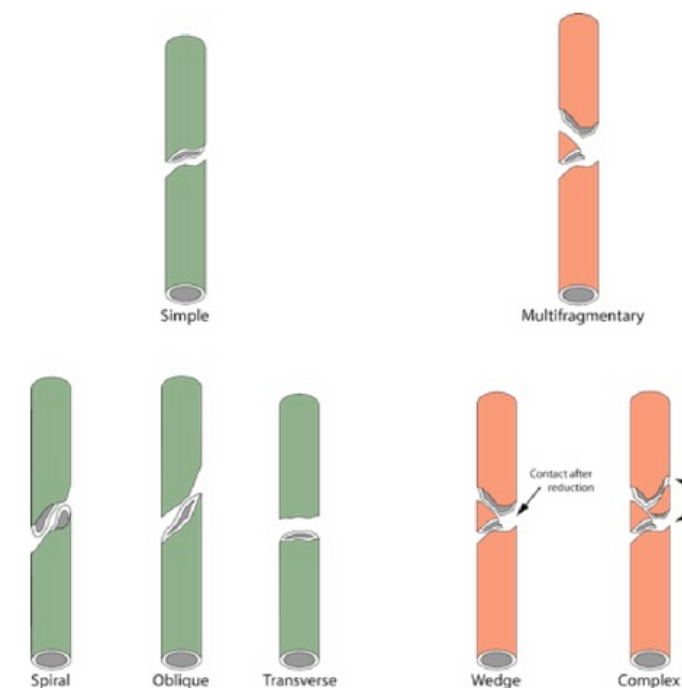
**Illustration 16.7** Type of fractures



INTERNATIONAL  
EMERGENCY  
MEDICINE  
EDUCATION  
PROJECT

# Ortho Pearls

## TYPES OF FRACTURES



Illustrated by Dejid Ahmetović

All fractures are either simple or multifragmentary (communited). A simple fracture (spiral, oblique or transverse), is a single circumferential disruption of any part of the diaphysis, metaphysis or articular surface. A multifragmentary fracture (communited) is any fracture with one or more completely separated fragments, which can be further classified as either wedge or complex. A wedge fracture consists of fragments that, after reduction, main fragments still have some contact between themselves; in a complex fracture, however, one or more intermediate fragments and main fragments are no longer in contact after reduction.



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## Description of Common Fractures

**Pathologic fractures:** A type of injury that results from a relatively small force applied to otherwise diseased or weakened bone, which in normal circumstances would not disrupt the cortex. Examples of such types of injuries are fractures through metastatic lesions, fractures through benign bone cyst and vertebral compression fractures in individuals with advanced osteoporosis.

**Stress fractures:** These types of fractures involve 'fatigued' bone tissue that was exposed to repetitive forces. The bone and supportive tissue did not have enough time to adequately accommodate such forces. A common example is the fracture of the metatarsal shaft in unconditioned foot soldiers and athletes. It is known as 'march fracture.'

**Salter-Harris fractures:** Fractures involving the physis and cartilaginous epiphyseal plate near the ends of the long bones in still growing children and

adolescents. Damage to the growth plate during growth may destroy part or all of its ability to produce new bone, thus preventing elongation of the bone, which may lead to anatomical and functional deformities.

Conveniently, the Salter-Harris fracture types can be memorized by the mnemonic **SALTR**.

S – (slipped), fracture plane passes all the way through the growth plate.  
A – (above), Fracture passes through most of the growth plate and up to the metaphysis.

L – (lower), A fracture that passes through the growth plate and extends down through the epiphysis.

T – (through, transverse or together), A fracture passing directly through metaphysis, growth plate and epiphysis.

R – (rammed, ruined), An uncommon crushing type of injury that does not displace the growth plate but damages it by direct compression.



## Illustration 16.8 SALTER-HARRIS Classification

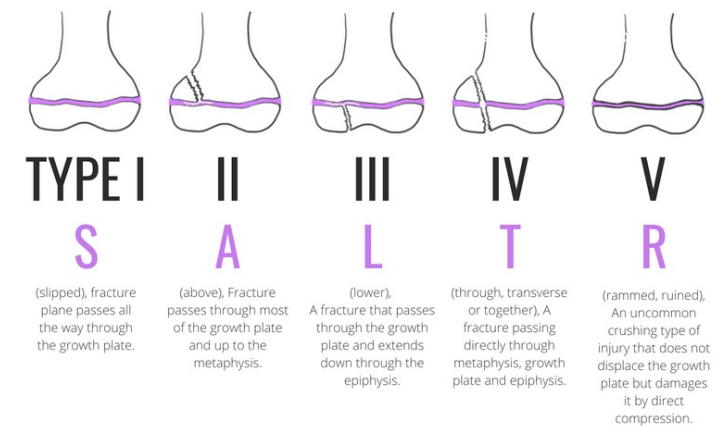


## Ortho Pearls

### SALTER - HARRIS CLASSIFICATION

Salter - Harris Fractures involve the physis and cartilaginous epiphyseal plate near the ends of the long bones in still growing children and adolescents. Damage to the growth plate during growth may destroy part or all of its ability to produce new bone, thus preventing elongation of the bone, which may lead to anatomical and functional deformities.

Salter-Harris fracture types can be memorized by the mnemonic SALTR.



Increase in Type of the fracture (I to V) increases the risk of complications.



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**Open fractures:** An open fracture is a fracture associated with overlying soft tissue injury, causing an open communication between the fracture or dislocation and the environment.

## Reduction

Reduction of fractures includes many options, some of which are appropriate

for one type of injury and some for another. The reduction can be either anatomical or non-anatomical.

Non-anatomical reduction in children is used for extra-articular fractures. Because of the remodeling potential in children, most deviations and anatomical positions will be corrected spontaneously, but only if no rotation is present. In adults, for example, fractures of the humeral shaft, deviations, and non-anatomical positions are well tolerated both functionally and cosmetically. The same applies to femoral and tibial shaft fractures when length, rotation, and axis remain the same.

Anatomical reduction in children is indicated in the case of some epiphyseal fractures, especially in those that are intra-articular because if the reduction is not perfect, the gap will be filled with callus, which can consequently cause premature closure of the growth plate. In intraarticular fractures in adults, the

reduction must be anatomical, or it could lead to joint incongruity and arthrosis.

### Treatment Options

General steps in fracture treatment are reduction, immobilization, and rehabilitation.

Conservative treatment involves either functional treatment or closed immobilization with or without any closed reduction. It is indicated in non-displaced fractures and when a certain degree of displacement is acceptable. Examples include clavicular, scapular and rib fractures, most stable vertebral types of fractures and pelvic fractures, also when the pelvis is stable, most extra-articular fractures in children because of their remodeling potential, and any extra-articular fractures when the anatomical position can be reached by closed reduction and maintained by closed external immobilization.

Surgical treatment involves open or closed reduction of the fracture and open

or closed fixation of the bone defect. Open reduction requires surgical intervention for alignment of the fracture fragments; however, in closed reduction, the fracture is reduced by manual manipulation of the affected area. There is also a difference between internal and external fixation. The term internal fixation itself suggests that the immobilizing implant is under the skin (bone surface or intramedullary), and external fixation presents in the case when most of the fixation material is outside the skin. (With this method, the risk of infection of the fracture is minimal. It is mostly used in severe open fractures.)

Generally, the indications for surgical treatment are open fractures, displaced intra-articular fractures, avulsion fractures and all femoral shaft fractures.

## Reductions of Selected Fractures

Any standard reduction procedure should include these steps

1. Confirming fracture with imaging if there is no neurovascular compromise and immediate reduction is needed
2. Defining the need of reduction procedure
3. Explaining the procedure to the patient and getting his/her consent for reduction and sedation and analgesia. You may also prefer to use hematoma or regional blocks.
4. Prepare the team and the equipment. Some fracture reductions may need more than one person if you are not using special traction devices for the reduction purpose. Prepare the post-reduction splinting/casting equipment as discussed in the [Splinting / Casting chapter](#).
5. Properly place the patient and injured extremity.
6. Properly position yourself and other team members.

7. Axial traction, rotation, or angulation maneuvers may be necessary for the different type of fractures. Therefore, apply proper technique accordingly.
8. After the reduction, please make sure the reduction is acceptable. Therefore, you can use imaging for the confirmation.
9. Stabilize the extremity as recommended in the [Splinting / Casting chapter](#).
10. If you are going to discharge the patient do not forget to give discharge instructions and arrange follow up with orthopedic clinic.

## Fracture of the middle phalanx

The mechanisms;

- direct force caused by fall,
- blow from a heavy object,
- twisting force.

**Image 16.18**



*Image shows comminuted fracture of the middle phalanx of 2nd finger.*

Pain, swelling, typical angulation because of extensor tendon are typical presentation.

Treatment is conservative in case of proper reduction. However often surgical fixation required. In general, simple axial traction is enough to align the fractured phalanx. However, keeping the fractured parts in an acceptable alignment can be



difficult. Therefore, immediate splinting/casting required.

## Boxer's fracture

Image 16.19



It is a fracture of the neck of the 4th. or 5th metacarpal. Image shows 5th metacarpal neck fracture. Swelling, pain and obvious deformity are seen in the presentation. Striking a clenched fist into an immovable object is the most common mechanism.

Treatment is conservative for acceptable angulation ( $30^\circ$  for 5th metacarpal) after reduction, surgical with severe displacement. The reduction requires

different specific maneuvers, please watch sample videos ([video 1](#) and [video 2](#) and [Video 3](#))

## Colles' fracture

Image 16.20



Fracture of the distal radius, with dorsal displacement and volar angulation.

Swelling and reduced movement and characteristic clinical deformity named 'dinner fork deformity' are seen in the presentation. It is the most common wrist

fracture in the elderly. Fall on an outstretched hand is the primary mechanism of injury.

Treatment options are conservative treatment with marginally displaced fractures and surgical (open reduction internal fixation (ORIF), external fixation) with severe displacement and unstable reduction.

Watch [video 4](#) and [video 5](#).

## Nightstick fracture

Image 16.21





It is a fracture of the shaft of either radius or ulna or both.

Deformity and pain are prominent in the presentation. The name of the fracture derived from citizen trying to defend against baton or nightstick, offering forearm. Caused by direct force, blow or impact.

Treatment is the conservative/closed reduction in stable and only slightly displaced fractures. Long arm cast with elbow 90°. The surgical option is ORIF with plate fixation in unstable fractures.

Forearm reduction [video](#).

## Fracture of the femoral shaft

**Image 16.22**



The figure shows a complex segmental fracture of the shaft.

Swelling, deformity, loss of function, pain, external rotation are presentation findings.

The mechanism is a direct or axial force of high energy. The reduction with axial traction should be applied in order to decrease pain, hemorrhage, and anatomical alignment. After the reduction, traction splint should be placed. The definitive treatment is surgical (ORIF) with plate or nail.

To learn how to apply Hare Traction Splint, please watch [video](#).

## Dislocations

Any standard reduction procedure should include these steps

1. Confirming dislocation with imaging if there is no neurovascular compromise and immediate reduction is needed
2. Defining the need for reduction procedure
3. Explaining the procedure to the patient and getting his/her consent for reduction and sedation and analgesia. You may also prefer to use the intraarticular anesthetic agent.
4. Prepare the team and the equipment. Many dislocation reductions may need more than one person if you are not using special traction devices for the reduction purpose. Prepare the post-reduction splinting/casting/sling equipment as discussed in the [Splinting / Casting chapter](#).

5. Properly place the patient and injured extremity.
6. Properly position yourself and other team members.
7. Axial traction, rotation, or angulation maneuvers may be necessary for the different type of dislocations. Therefore, apply proper technique accordingly.
8. After the reduction, please make sure the joint is in normal anatomy. Therefore, you can use imaging for the confirmation.
9. Stabilize the extremity as recommended in the [Splinting / Casting chapter](#).
10. If you are going to discharge the patient do not forget to give discharge instructions and arrange a follow up with the orthopedic clinic.

## Reductions of Selected Dislocations

### Dislocation of the interphalangeal joints of the fingers

Image 16.23



*The image shows a fracture and subluxation at the distal and fracture and dislocation at the proximal phalangeal joint.*

Dislocations of the PIP joint are a common hand injury, as opposed to DIP dislocation, which is rare, because of the firm attachments of the skin and surrounding tissue to the bone.

Pain, deformity are two main characteristics at the presentation.

Axial loading and hyperextension cause the dislocation. Dislocations are usually dorsal.

Longitudinal traction and hyperextension with applying dorsal pressure to the base of the dislocated phalanx usually reduce the dislocation.

Finger dislocation reduction [video](#).

Finger dislocation metacarpal block [video](#).

### Shoulder dislocation

Image 16.24



*The images show loss of shoulder curve on the left, and X-ray of the same patient with anterior shoulder dislocation and severe Hill Sacks deformity (cortical depression in the posterolateral head of the humerus) + fracture.*

Anterior glenohumeral dislocation, the most common type of shoulder dislocation. Pain and swelling are common, and normal rounded contour of the shoulder is lost. The patient is unable to move, supported with healthy arm. The mechanism is generally a fall on the hand when in the externally rotated position. It is common in age 18-25, mostly due to sport or motorbike injury.

The majority of the cases are successfully reduced with simple maneuvers (conservative reduction). Traction and external rotation-elevation, scapular rotation, Cunningham are the most popular techniques. However, there are many other successful methods. Please do not use the Hippocrates and Kocher techniques because of their high complication rate. If the reduction is unsuccessful, it can be done under sedation or even in general anesthesia in some cases.

An error occurred.

Try watching this video on [www.youtube.com](http://www.youtube.com), or enable JavaScript if it is disabled in your browser.

10 ways to reduce dislocated shoulder - [video](#).

Cunningham technique to reduce shoulder dislocation - [video](#).

## Hip dislocation

**Image 16.25**



*The images show posterior hip dislocation.*

In approximately 95% femoral head is dislocated posteriorly.

In the posterior dislocation, the leg position is in flexion, adduction and internal rotation.

In the anterior dislocation, the leg is in extension, abduction and external rotation.

High-energy trauma with the flexed knee (e.g., dashboard injury).

Conservative reduction is necessary, if unsuccessful, reoccurring or any type of acetabular injury, surgical treatment is required.

Watch the [video](#) for reduction of the hip.

Artificial hip dislocation - reduction - [video](#).

**References and Further Reading**, click [here](#)

# Splinting and Casting

by Joseph Pinero, Timothy Snow, Suzanne Bentley

## Case Presentation 1

*65-year-old female with a history of hypertension and diabetes presenting with right wrist pain and swelling after suffering a fall from standing, landing on an outstretched hand. Plain radiographs of the wrist will show a distal radius fracture, otherwise known as a Colles fracture.*

Image 16.26



## Procedure: Sugar Tong

Emergency Indication

- Colles Fracture (Distal Radius Fracture)
- Distal Ulna Fracture
- Smith Fracture (Reverse Colles Fracture)



- Barton Fracture (Dorsal or volar rim fracture of the distal radius)

### Precaution

The presence of an open fracture requires an emergent orthopedic surgical consultation and surgical fixation with open reduction and internal fixation.

### Equipment and Patient Preparation

- Syringe
- Lidocaine
- 4" Plaster
- Soft web roll lining
- 4" ace wrap
- Sling

### Procedure Steps

1. Obtain standard radiographs including posteroanterior and lateral films
2. Local anesthesia via hematoma block

- Contraindicated if overlying cellulitis or grossly contaminated wound
- Clean skin with an antiseptic solution
- First, anesthetize skin with a small wheal of lidocaine
- Then dive deeper into the largest area of swelling and hematoma and aspirate blood. Once confirmed that you are within the hematoma, gently inject 10-15 cc of anesthetic.
- Wait at least 10 minutes for the anesthetic to be absorbed prior to beginning your manipulation.

3. Perform a closed reduction with the aim of creating a neutral volar tilt (15-degree angulation in wrist flexion)
  - The sugar tong splint should be applied by placing a U-shaped splint from the dorsal metacarpal-phalangeal joints down around the elbow joint and wrap back around to come up to just below the metacarpal-phalangeal joints on the palmar surface.

- The plaster should be measured prior to placement and should be 8-10 layers thick

4. Place the patient in a sling and perform post-reduction plain radiographs

Please watch the [video](#).

### Hints and Pitfalls

- Using too much padding can cause your splint not to provide enough support, resulting in malunion.
- Using too little padding can result in plaster burning the skin.

### Post Procedure Care and Recommendations

Always take post-reduction radiographs

### Complications

- Malunion
- Nonunion
- Median nerve injury

## Case Presentation 2

*11-year-old male with no past medical history presents with right arm pain around the elbow after falling off of the monkey bars at the playground earlier today. Plain radiographic films will show a supracondylar fracture.*

**Image 16.27**



## Procedure: Posterior Long Arm Splint

Emergency Indication

- Supracondylar fracture
- Distal Humerus fracture
- Monteggia's fracture
- Proximal forearm fractures
- Radial head and neck fractures
- Olecranon fractures
- Severe ligamentous injuries of the elbow

### Precaution

The presence of an open fracture requires an emergent orthopedic surgical consultation and surgical fixation with open reduction and internal fixation

### Equipment and Patient Preparation

- Syringe
- Lidocaine

•4" Plaster

- Soft web roll lining
- 4" ace wrap
- Sling

### Procedure Steps

1. Obtain standard radiographs including posteroanterior and lateral films
2. Local anesthesia is typically not effective in this group as these fractures are typically seen in young children that require minimal manipulation if any at all.
3. If a child cannot tolerate the placement of the splint, it is possible to utilize procedural sedation (administration of a small amount of sedative making the child less aware of the procedure).
4. Placing the splint requires pre-measurement of plaster casting material from the proximal posterior humerus at the axillary crease to the

wrist joint without crossing into the hand.

- The plaster should be layered at 8-10 layers thick, as this is a long arm splint and will be heavier than the average short arm splint.
- The elbow should be placed at 90 degrees of flexion with the wrist in a neutral position, which is neither supinated nor pronated.

5. Place the patient in a sling and perform post-reduction plain radiographs.

Hints and Pitfalls

6. Be sure to place the arm in the proper position of neutrality.

Post Procedure care and recommendations

7. Make sure to provide adequate analgesia both before and after the placement of the splint.

Please watch the [video](#).

## Complications

- Malunion
- Nonunion
- Median nerve injury
- Pressure ulcers
- Decreased range of motion

## Case Presentation 3

*26-year-old male with no past medical history presents with left ankle pain after landing on another player's foot while jumping up during a basketball game.*

Image 16.28



## Procedure: Short Leg Splint

### Emergency Indication

- Ankle fracture
- Tibia fracture
- Severe ankle sprain
- Metatarsal fractures

### Precaution

The presence of an open fracture requires an emergent orthopedic surgical consultation and surgical fixation with open reduction and internal fixation.

### Equipment and Patient Preparation

- Chucks (or any material that you can use to keep the counters and floors clean)
- Water source (basin half full of water will suffice)
- Syringe
- Lidocaine

- 4" or 6" Plaster (depending on the size of the leg)
- Soft web roll lining
- 6" ace wrap
- Crutches

### Procedure Steps

1. Obtain standard radiographs including posteroanterior and lateral films
2. Local Anesthesia via hematoma block
  - Contraindicated if overlying cellulitis or grossly contaminated wound
  - Clean the skin with an antiseptic solution
  - First, anesthetize skin with a small wheal of lidocaine
  - Then dive deeper into the wound overlying the largest area of swelling and hematoma and aspirate blood. Once confirmed that you are within the hematoma, gently inject 10-15 cc of anesthetic.

- Wait at least 10 minutes for the anesthetic to be absorbed prior to beginning your manipulation.

3. Placing a short leg splint for this type of fracture involves pre-measuring two separate strips of plaster.

- The first strip is measured from the superior calf to just beyond the toes of the foot for your posterior support
- The second strip is measured from the head of the fibula, down and around the heel, up to the tibial plateau medially.
- Once both strips are measured, wrap the leg in web roll padding and place the plaster on the leg with the U-shaped splint above the posterior support strip.
- Cover both with a thin layer of the web roll, followed by your ace bandage.
- The leg will typically require more than one 6" ace wrap



Perform post-reduction plain radiographs and educate the patient on the use of crutches

Please watch [video](#).

### Hints and Pitfalls

Test for neurovascular function

### Post Procedure care and recommendations

Make sure to provide adequate analgesia both before and after the placement of the splint.

### Complications

- Malunion
- Nonunion
- Pressure ulcers
- Decreased range of motion
- Early onset arthritis

## Case Presentation 4

*A 8-year-old male presenting with thigh pain after falling from a bicycle. Plain radiographs show a fracture of the distal femur.*

Image 16.29



## Procedure: Long Leg Splint

### Emergency Indication

- Distal femur fracture
- Tibia plateau fracture

### Precaution

The presence of an open fracture requires an emergent orthopedic surgical consultation and surgical fixation with open reduction and internal fixation.

### Equipment and Patient Preparation

- Chucks (or any material that you can use to keep the counters and floors clean)
- Water source (basin half full of water will suffice)
- Syringe
- Lidocaine

Please watch [video](#).

- 4" or 6" Plaster (depending on the size of the leg)
- Soft web roll lining
- 6" ace wrap
- Crutches

### Procedure Steps

1. Obtain standard radiographs including posteroanterior and lateral films
2. Local Anesthesia via hematoma block
  - Contraindicated if overlying cellulitis or grossly contaminated wound
  - Clean skin with an antiseptic solution
  - First, anesthetize skin with a small wheal of lidocaine
  - Then dive deeper into the wound overlying the largest area of swelling and hematoma and aspirate blood. Once confirmed that you are within the hematoma, gently inject 10-15 cc of anesthetic.

- Wait at least 10 minutes for the anesthetic to be absorbed prior to beginning your manipulation.

3. Placing a long leg splint for this type of fracture involves pre-measuring three strips of plaster: one from the plantar surface of the toes to the gluteal fold, one support strut from the medial ankle up to the proximal inner thigh, and the last support strut from the lateral ankle up to the greater trochanter of the femur.
  4. Wrap the leg in web roll padding
  5. Firmly secure the splint with a top layer of web roll and ace wrap
- Hints and Pitfalls
6. Test for neurovascular function
  7. If the fracture is in the midshaft of the femur or proximal femur, casting is not an appropriate option. Orthopedic consultation and traction fixation will be required temporarily, prior to surgical fixation.

### Post Procedure Care and Recommendations

Make sure to provide adequate analgesia both before and after the placement of the splint.

### Complications

- Malunion
- Nonunion
- Pressure ulcers
- Decreased range of motion
- Early onset arthritis

## Case Presentation 5

54-year-old female with no past medical history presents with right ankle pain after stepping off of a curb and “rolling over her ankle.” X-ray shows a non-displaced malleolar fracture.

Image 16.30



## Procedure: Short Leg Cast

### Emergency Indication

- Definitive treatment for nondisplaced ankle and foot fractures
- The presence of an open fracture requires an emergent orthopedic surgical consultation and surgical fixation with open reduction and internal fixation.

### Equipment and Patient Preparation

- Chucks (or any material that you can use to keep the counters and floors clean)
- Water source (basin half full of water will suffice), lukewarm temperature
- Syringe
- Lidocaine
- 4” and 6” Plaster (depending on the size of the leg)

• Soft web roll lining

- 6” ace wrap X 2
- Crutches

### Procedure Steps

1. Obtain standard radiographs including posteroanterior and lateral films
2. Local anesthesia via hematoma block, and for more severe fractures, systemic analgesia may be required.
  - Contraindicated if overlying cellulitis or grossly contaminated wound
  - Clean the skin with an antiseptic solution
  - First, anesthetize skin with a small wheal of lidocaine
  - Then dive deeper into the wound overlying the largest area of swelling and hematoma and aspirate blood. Once confirmed that you are within the hematoma, gently inject 10-15 cc of anesthetic.

- Wait at least 10 minutes for the anesthetic to be absorbed prior to manipulation.

3. Placing a short leg cast for this type of fracture involves first ensuring the foot is sitting in proper anatomical alignment ankle flexed at 90 degrees.

- The first step is to adequately pad the entire area of casting (from 1-2cm distal to the tibial plateau) to the distal foot (covering the base of the phalanges) leaving the tips of the toes uncovered. Extra padding should be placed at the areas of pressure (the ends of the cast) and the heel to prevent ulcers.

- The second step is to apply the plaster ensuring the ankle remains at 90 degrees of flexion. The entire rolls of plaster are dipped and soaked in lukewarm water and then squeezed to remove some of the water. The thumb and index finger should be placed at each end of the plaster to

prevent “bananaing” of the plaster when applying.

- Plaster or fiberglass is then wrapped around the foot and ankle up to the proximal tibia ensuring it remains in 90 degrees of flexion.
- A foot plate can be added by placing 6 layers of plaster on the bottom of them for support.
- To prevent cutting off blood flow to the distal foot when the injury swells, bi-valving of the cast should be done by cutting along the medial and lateral shin, through the plaster or fiberglass.

4. Give the patient crutches and perform post-reduction plain radiographs

Please watch [video](#).

### Hints and Pitfalls

- Extra padding should be applied to areas of pressure (tips of toes, heel at the malleoli and top of the cast at the

tibial plateau) to prevent pressure ulcers.

- Test for neurovascular function after casting
- Short leg casts are often “bi-valved” or cut in half prior to discharge from the hospital. This is done in order to allow for some room for swelling.

### Post Procedure Care and Recommendations

Make sure to provide adequate analgesia both before and after the placement of the splint.

### Complications

- Malunion
- Nonunion
- Pressure ulcers
- Decreased range of motion
- Early onset arthritis



- Contracture of the Achilles tendon if the foot is <90 degrees of flexion

**References and Further Reading**, click [here](#)

# Urinary Catheter Placement

---

by Gul Pamucu Gunaydin

## Case Presentation

*A 75-year-old male patient was admitted to the emergency department with difficulty voiding. He had this complaint for over a year, and tonight, although he felt pain and distention in his lower abdomen, he could not urinate at all. On his physical exam, the patient had a palpable mass that was thought to be the distended bladder. He was agitated and tachycardic. He was diagnosed with acute urinary retention, and initial attempt to insert urinary indwelling catheter was failed. The second attempt with a Coude catheter was successful and 2 liters of urine was drained gradually. His rectal exam revealed prostate enlargement. He was discharged with instructions, uneventfully.*

## Procedure: Urinary Catheter Placement

Urinary catheter insertions is a common procedure in the ED. They may be external (condom) or indwelling (urethral, suprapubic). Condom catheters are indicated in men with functional disabilities such as restricted mobility or dementia

with incontinence, who can void spontaneously. Suprapubic catheters are an option if urethral catheters fail. This chapter focuses solely on urethral urinary catheterization.

## Emergency Indications

### Short-term catheterization

#### Diagnostic

- Diagnostic sampling (sterile urine sampling)
- Monitoring urinary output (trauma, critically ill, burns)
- Filling the bladder prior to pelvic ultrasound
- Cystogram, cystourethrogram
- Urine collection
- Monitoring core body temperature

#### Therapeutic

- Draining urine in acute urinary retention, urinary obstruction, inability to void
- Irrigation of bladder to remove gross hematuria and clots/debris
- Palliative care for terminally ill (e.g. to assist treatment of decubitus ulcers in incontinent patients by maintaining moisture free environment)
- To warm hypothermic patients
- Intubated patient
- Emergency Surgery

### Long-term catheterization

- Bladder outlet obstruction
- To reduce changes in patients who are terminally ill or cannot care for themselves
- Neurogenic bladder
- Urinary incontinence

### Contraindications

#### Absolute

- Trauma patient presenting with the following signs (known or suspected urethral damage):
  - Blood at meatus
  - Penile deformity
  - High riding prostate
  - Perineal hematoma
- Allergy to latex, rubber or lubricants

#### Relative

- Uncooperative patient
- Recent bladder or urethral surgery
- Urethral Stricture

### Equipment and Patient Preparation

Urinary catheter: Catheters are classified according to the material it is made of, number of lumens and shape of the tip.

Number of lumens

- One way-non balloon also known as straight, Nelaton or Robinson catheters are used for one time or intermittent drainage.
- Two-way catheters have a balloon inflation channel and a urine drainage channel.
- Foley catheter, which has a self-retaining balloon, is the most commonly used.
- The triple lumen (three-way) indwelling catheter is used for bladder irrigation.

#### Shape of Tip

- Coude or Tieman catheter curves 45 degrees at the tip and is designed to pass urethra in patients with prostatic enlargement; it offers rigidity too.
- The Whistle Tip (Couvelaire Tip) catheter has a terminal and a lateral drainage eye used for large blood clots.

- The Roberts tip catheter has an eye above and below the balloon to reduce the residual urine.

Catheter size is described in French units. It refers to the catheter's circumference in millimeters. Start with 12-16 F for adults. Choose the smallest size that is enough for adequate drainage. If obstruction of the catheter due to blood or debris is expected, use a larger bore catheter (e.g., 18-24 F).

Catheter length: Adult indwelling catheters are available in a standard (male) length (40-45cm) and a shorter female length (20-26cm). Female length catheters should not be used in male patients because of the risk of inflating the balloon in the urethra.

#### Other Equipment:

- Sterile gloves and drapes
- Sterile gauze sponge or cotton balls
- Antiseptic solution (Povidone-iodine or chlorhexidine)

• Sterile local anesthetic lubricant gel: (% 2 lidocaine gel) anesthetizing the urethra with topical lidocaine gel instilled through a pre-loaded syringe reduces discomfort. The catheter tip is also lubricated prior to its insertion.

- 10 ml syringe filled with sterile saline or sterile water
- Sterile urine bag
- Tape to secure the urine collection system

## Procedure Steps

Universal precautions should be taken in all steps. Patient consent should be obtained before starting any procedure. Ensure the privacy of the patient. Aseptic insertion technique is recommended.

## Female Patients

1. Prepare all equipment on a tray covered with a sterile drape in a sterile fashion.
2. Place the patient in the lithotomy position.



3. Wear your sterile gloves.
4. Check the balloon for patency.
5. Place a fenestrated drape over the perineum.
6. Spread the labia with your non-dominant hand.
7. Use the forceps/pickups to hold the sterile sponge, soak it in the antiseptic solution, and clean the area from anterior to posterior and central to peripheral.
8. Alternatively, you may change gloves after cleansing external genitals.
9. Lubricate the tip of the catheter with %2 lidocaine gel.
10. Pass the catheter through the meatus and advance it until the hub meets the urethral meatus, you should be able to see urine flowing. Insert the catheter 2-3 inch or 5-7.5 cm more, preferably until the hub to avoid inflating the balloon inside the urethra.

11. Inflate the balloon with 10 ml of sterile water or saline using the filling port.
12. Pull the catheter back until resistance is felt.
13. Attach the urine collection bag.
14. Secure the catheter to the anterior thigh.
15. Remove gloves, dispose of waste appropriately, and wash hands.

Please watch below videos ([manikin](#) and [patient](#) examples)

### Male Patients

Perform step 1 to 4 of female patient catheterization.

5. Firmly hold the penis with the non-dominant hand, and position the penis 45 to 90 degrees to the coronal plane, apply gentle traction. Retract the foreskin if the patient is not circumcised.
6. Use the forceps/pickups to hold the sterile sponge, soak it in antiseptic

solution and paint the area in a sterile fashion with the antiseptic solution.

7. Alternatively, you may change gloves after cleansing external genitals.
8. Inject 10 mL of 2% lidocaine gel into the urethra through the meatus before insertion of the catheter.
9. Perform step 10-15 of female catheterization.
10. When the procedure is finished, don't forget to reduce foreskin to prevent iatrogenic paraphimosis.

Please watch below videos ([manikin](#) and [patient](#) examples)

### Hints and Pitfalls

- Universal availability and ease of insertion of urinary catheters often lead to the inappropriate and prolonged use of these catheters. Insert catheters only for appropriate indications and leave catheters in place only as long as needed.

- A tense patient means a tight urethral sphincter; encourage the patients to relax by taking deep breaths and relax urinary sphincter muscles as if going to void.
- Always be gentle; never force the catheter since this may cause urethral trauma.
- If no urine has returned, do not inflate the balloon.
- Even when urine is flowing, it is possible for the eye of the catheter to lie within the bladder while the balloon remains within the prostatic urethra; so, always advance the catheter until the hub.
- If there is pain during inflation of the balloon, stop immediately since the balloon may still be in the urethra.
- Once inserted, the catheter should be secured to prevent traction and damage from movement and catheter kinks.

- Place the urinary drainage bag below the level of the patient's bladder, not allowing it to touch the floor.
- For difficult urinary catheterization, change the size: 20-24 F catheter for benign prostate hyperplasia, small caliber for the urethral stricture (12-16 F).
- If catheterization is unsuccessful, it is best to avoid multiple blind attempts since they increase the risk of infection, exacerbate the patient's discomfort, and produce urethral congestion and edema, rendering further attempts even more challenging.
- Patients occasionally experience hypotension and hematuria when the large volume from the bladder is drained rapidly but has little clinical significance, and gradual emptying is not necessary.

## Post-Procedure Care and Recommendations

• Patients' follow up with urology should be arranged.

### • Discharge instructions:

- If you develop any symptoms of a urinary tract infection, contact your doctor immediately.
- Take enough fluids to maintain adequate urine flow.
- Be careful not to pull the catheter accidentally, avoid twisting and kinking of the catheter.
- Keep the bag lower than the bladder to prevent back flowing.
- Avoid disconnecting the catheter and drain tube.
- Empty the bag regularly. The drainage spout should not touch anything while emptying the bag.
- Alpha blockers may be started to patients with prostate enlargement.

## Complications

- Discomfort, pain
- Inability to pass the catheter
- Misplacement of the catheter
  - Vagina
  - Ureter
  - Renal Pelvis
- Traumatic complications to lower urinary tract – proper insertion technique is the single most important factor for preventing injury.
  - Passage of the catheter into a false lumen
  - Intraurethral balloon distention
  - Hematuria
  - Rupture of urethra 11 (may cause urethral stricture in the long term) 5
  - Bladder perforation
  - Hydro uterus
  - Paraphimosis

- Vena cava air embolism
- Infections: UTI accounts for 32% of all healthcare-associated infections. A majority of these infections are attributable to the use of an indwelling catheter. Use of best practice techniques by emergency nurses can help prevent UTIs from occurring as a result of urinary catheter insertions in the emergency department. Earlier catheter removals, use of smaller bore catheters, a closed drainage system, optimal hygienic techniques (hand-washing, sterile catheterization techniques) by health care workers, and removal of the catheter when infection is suspected are effective in minimizing the incidence of infection.
  - Urinary tract infection
    - Urethritis
    - Prostatitis
    - Epididymo-orchitis
    - Cystitis

- Pyelonephritis
  - Bacteremia, urosepsis
- Latex allergies
- Obstruction or blockage of catheter results from precipitated mucus, protein, crystals, blood clots, and bacteria.
- Urine leakage around the catheter
  - Fragmentation or fracture and retainment of the catheter
  - Catheter knotting
  - Balloon rupture
  - Calculi formation
  - Bladder spasms contraction
  - Accidental removal of the catheter
  - Stricture formation in long-term

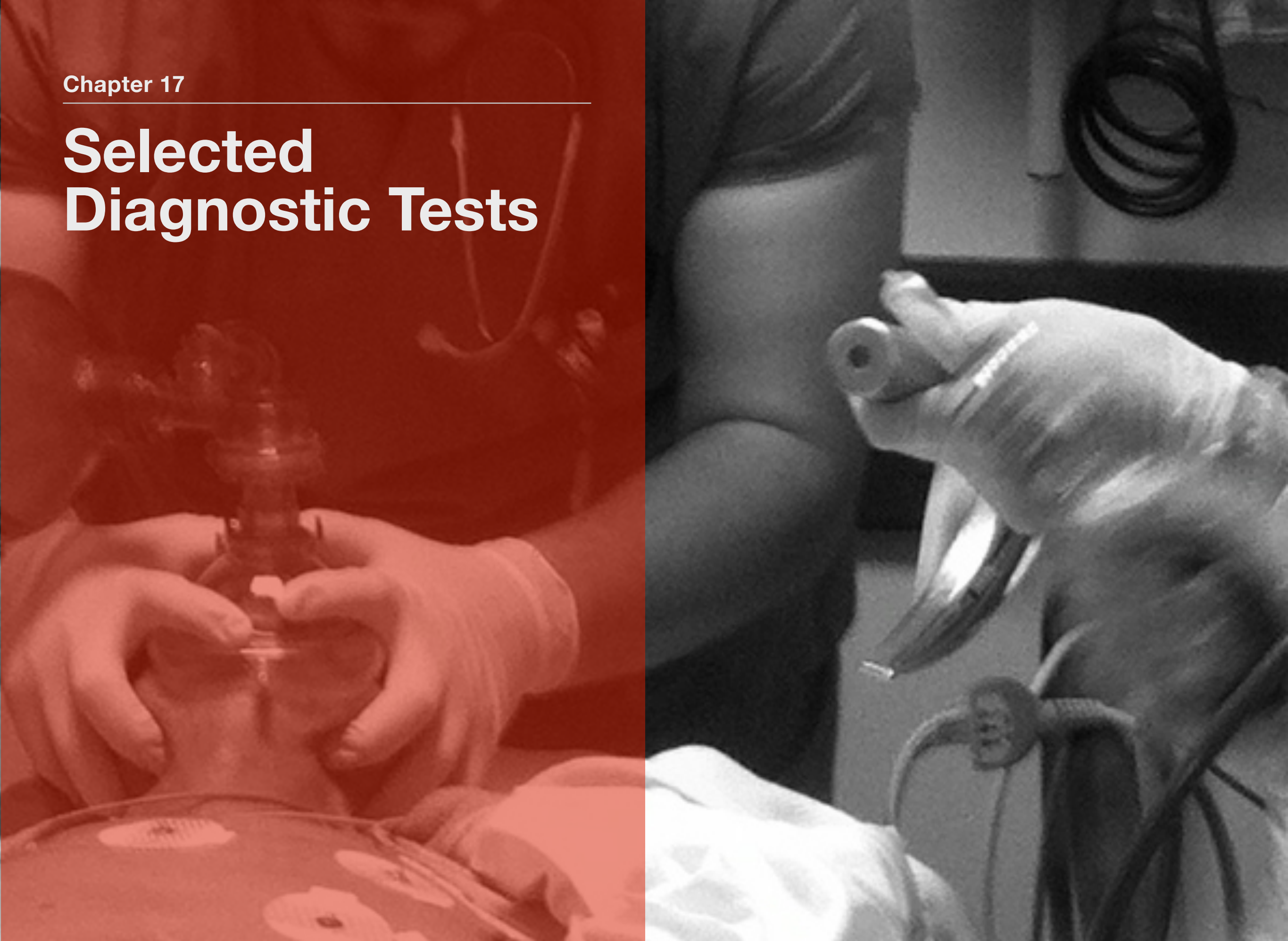
## Pediatric, Geriatric, Pregnant Patients and Other Considerations

- Use 6-10 F catheters for pediatric patients, 12F for patients age >12 years, 5F for infants
- Difficult urethral catheterization (DUC) is where the urological consult is requested to insert a urinary catheter. Many causes of DUC have been identified including anxiety, poor technique, urethral stricture, phimosis, bladder neck contracture, false passages, benign prostatic hyperplasia, unfavorable body habitus and patient positioning.
- To prevent infections:
  - Insert catheters using aseptic technique and sterile equipment
  - Maintain a closed drainage system
  - Maintain unobstructed urine flow

**References and Further Reading**, click [here](#)



# Selected Diagnostic Tests



# Arterial and Venous Blood Gas Analysis

by Kemal Gunaydin

## Introduction

Measurements of  $\text{PaO}_2$ ,  $\text{PaCO}_2$ ,  $\text{SaO}_2$ , pH, and bicarbonate values are made with arterial blood gas (ABG) analysis in order to determine the acid-base balance and respiratory regulation. Arterial blood gas (ABG) analysis is an important laboratory method that provides reliable information about the patient's metabolic status and respiratory physiology.

## Indications for arterial blood gas (ABG) analysis are

- Diagnosis and follow-up of metabolic and respiratory acidosis and alkalosis
- Determination of the type of respiratory failure

- Determination of the need for mechanical ventilation
- Evaluation of the indication for admission to intensive care
- Determination of the effectiveness of the given treatment
- Indication and follow-up of oxygen treatment
- Evaluation of the reason for sudden and unexplained dyspnea

Generally, the radial, brachial and the femoral arteries are used for this purpose. The choice of the artery is associated with many factors. It mainly depends on the physician's experience and the patient's clinical condition. Primarily, the radial artery is preferred. The Allen test should be performed

prior to the procedure to evaluate the adequacy of the collateral circulation in hand. The obtained blood gas sample should be delivered to the laboratory as soon as possible.

The normal values of the arterial blood gases (Please refer to the agreed norms from your lab);

pH 7.35 – 7.45

PaCO<sub>2</sub> 35 – 45 mmHg

PaO<sub>2</sub> 80 – 100 mmHg

SaO<sub>2</sub> %95 – 97

Standard HCO<sub>3</sub> 22 – 26 mEq/L

Actual HCO<sub>3</sub> 22 – 26 mEq/L

BE (Base excess) ±3 mmol/L

[H<sup>+</sup>]: Hydrogen ion concentration.

pH: The negative logarithm of the hydrogen ion concentration.

PaO<sub>2</sub>: Oxygenation

PaCO<sub>2</sub>: Alveolar ventilation

PaO<sub>2</sub> and PCO<sub>2</sub>: Gas exchange

Ph, PCO<sub>2</sub>, and HCO<sub>3</sub>: These are used to evaluate the acid-base status.

## pH

In ABG, pH shows a status of acidosis or alkalosis. However, it is not possible to understand its type with pH. pH is also the only parameter showing compensation. Its normal values are between 7.35-7.45. It is decompensated acidosis if pH<7.35, and decompensated alkalosis if pH>7.45.

## Arterial partial pressure of oxygen (PaO<sub>2</sub>)

This is the partial pressure of oxygen in the arterial blood. It is used in the evaluation of oxygenation.

- PaO<sub>2</sub>: Between 60-79 mm Hg, “mild hypoxemia.”

- PaO<sub>2</sub>: Between 40-59 mm Hg, “moderate hypoxemia.”

- PaO<sub>2</sub>: Below 40 mmHg, “severe hypoxemia.”

## Arterial partial pressure of carbon dioxide (PaCO<sub>2</sub>)

This is the partial pressure of carbon dioxide in the arterial blood. It is the indicator of alveolar ventilation. Its normal value is 40 mmHg at sea level, while it is 46.5 mmHg in venous blood. Increased values show respiratory acidosis, while decreased values demonstrate respiratory alkalosis.

## Alveolar-arterial oxygen gradient – p(A-a) O<sub>2</sub>

This is the difference between the alveolar and arterial partial pressures of oxygen, providing general information about the function of gas exchange in the lungs. Its normal value is 5 mmHg, which increases with age. A 4 mmHg increase is observed for every 10 years after 20 years of age.



$p(A-a) O_2: [150 - (1.25 \times PaCO_2)] - PaO_2$

Expected  $p(A-a) O_2$  value for age:  $2.5 + [0.25 \times \text{age}(\text{years})]$

## Bicarbonate ( $HCO_3^-$ )

This is the serum concentration of the bicarbonate ion. It is an important buffer in the blood, and it is used to evaluate the metabolic component of the acid-base balance. Standard bicarbonate is the bicarbonate value that should be present in the blood under standard conditions ( $37^\circ\text{C}$  temperature and 40 mmHg  $PCO_2$ ). Its normal value is 22-26 mEq/L. Actual bicarbonate is the real bicarbonate value in the blood. Its normal value is 22-26 mEq/L. Increased values indicate metabolic alkalosis, while decreased values show metabolic acidosis.

## Base excess (BE)

Metabolic acidosis or alkalosis may be determined by looking at the base excess. BE is the amount of required acid or base to bring the pH of the totally oxygenated blood to 7.40 at  $37^\circ\text{C}$  and 40 mmHg  $PCO_2$ ; it is the indicator of the

metabolic status. If BE is  $< -2.5$ , it is metabolic acidosis, if BE  $> +2.5$ , it is metabolic alkalosis.

## Anion Gap (AG)

The anion gap represents the difference between the serum cations and the anions. In daily practice, the measured cation is sodium, and the anions are chloride and the bicarbonate. The normal AG is  $12 \pm 4$  mEq/L. Albumin constitutes the majority of the immeasurable anions. In patients with low levels of albumin, AG should be considered according to the level of albumin. It shows whether the metabolic acidosis develops due to the accumulation of non-volatile acids (lactic acid, ketoacids, etc.) (increased AG metabolic acidosis), or due to loss of bicarbonate (normal AG or hyperchloremic metabolic acidosis).

$$AG = Na^+ - (HCO_3^- + Cl^-)$$

Expected AG = Calculated AG +  $2.5 \times [4.5 - \text{albumin level}]$

## Delta-Delta Gap ( $\Delta AG / \Delta HCO_3^-$ )

In the presence of high AG metabolic acidosis, the “delta-delta gap” is calculated to determine a second metabolic acid-base balance imbalance. In this case, the increase in AG is compared with the decrease in  $HCO_3^-$ .  $AG / \Delta HCO_3^- = (\text{Calculated AG} - 12) / (24 - \text{measured } HCO_3^-)$

- In the presence of high AG metabolic acidosis,  $\Delta AG / \Delta HCO_3^- = 1$ .
- If there is also hyperchloremic acidosis,  $\Delta AG / \Delta HCO_3^- < 1$ .
- If there is also metabolic alkalosis,  $\Delta AG / \Delta HCO_3^- > 1$ .

## Lactate

Lactate is a surrogate anaerobic indicator of metabolism, which is increased under stress and hypoperfusion. It is also used as an indicator of the resuscitative efforts in patients with shock and an indicator of survival in patients with septic shock.



Levels above 4 mmol/L are associated with a mortality rate of 28%.

## SYSTEMATIC INTERPRETATION OF THE ARTERIAL BLOOD GASES

### 1th STEP

If pH or PaCO<sub>2</sub> are out of normal range, acid-base balance imbalance is present

### 2th STEP

If pH is abnormal, and pH and PaCO<sub>2</sub> move in opposite directions, the primary disorder is RESPIRATORY

If pH is abnormal, and pH and PaCO<sub>2</sub> move in the same direction, the primary disorder is METABOLIC

### 3th STEP

If one of pH or PaCO<sub>2</sub> is normal, a mixed acid-base disorder is present.

If pH is normal, the change in the direction of PaCO<sub>2</sub> defines a respiratory disorder.

- If PCO<sub>2</sub> ↑ = Respiratory acidosis-metabolic alkalosis

- If PCO<sub>2</sub> ↓ = Respiratory alkalosis-metabolic acidosis

If PaCO<sub>2</sub> is normal, the change in the direction of pH defines a metabolic disorder.

- If pH ↑ = Metabolic alkalosis-respiratory acidosis
- If pH ↓ = Metabolic acidosis-respiratory alkalosis

### 4th STEP

If primary metabolic acidosis or alkalosis is detected, the expected PaCO<sub>2</sub> is calculated.

- For metabolic acidosis;
  - Expected PaCO<sub>2</sub> = (1.5 x HCO<sub>3</sub>) + 8 ± 2
- For metabolic alkalosis;
  - Expected PaCO<sub>2</sub> = 40 + (0.6 x ▲HCO<sub>3</sub>)

If PaCO<sub>2</sub> is within the expected range, full compensation is present.

If it is more than the expected value, concomitant respiratory acidosis is present.

If it is less than the expected value, concomitant respiratory alkalosis is present.

### 5th STEP

If there is respiratory acidosis or alkalosis present, the expected pH is calculated.

In acute respiratory acidosis;

- Expected pH = 7.4 - [ 0.008 x (PaCO<sub>2</sub>-40)]

Chronic respiratory acidosis;

- Expected pH = 7.4 - [ 0.003 x (PaCO<sub>2</sub>-40)]

If pH is below the expected value in acute respiratory acidosis, there is concomitant metabolic acidosis present.

If it is above the expected value in chronic respiratory acidosis, there is concomitant metabolic alkalosis present.

In acute respiratory alkalosis;

- Expected pH =  $7.4 + [0.008 \times (40 - \text{PaCO}_2)]$

In chronic respiratory alkalosis;

- Expected pH =  $7.4 + [0.003 \times (40 - \text{PaCO}_2)]$

If pH is above the expected value in acute respiratory alkalosis, there is concomitant metabolic alkalosis present.

If it is below the expected value in chronic respiratory alkalosis, there is concomitant metabolic acidosis present.

## 6TH STEP

The evaluation of the anion gap in metabolic acidosis

### Acid-base disorders

#### Metabolic

Causes of Acidosis with Increased Anion Gap

- Lactic Acidosis

- Ketoacidosis
- Uremic Acidosis
- Methanol poisoning
- Ethanol poisoning
- Ethylene Glycol poisoning
- Propyl Alcohol Poisoning
- Salicylate Poisoning
- Iron Poisoning

Causes of Acidosis with Normal Anion Gap

- Diarrhea
- Isotonic Saline Infusion
- Renal failure
- Renal tubular acidosis
- Acetazolamide
- Ureteroenterostomy

Causes of Alkalosis with Decreased Amount of Fluid

- Gastric acid loss
  - Vomiting
  - Gastric aspiration
- Renal Cl loss
  - Use of diuretics
  - Urine Cl < 20mmol/L

Causes of Alkalosis with Normal Amount of Fluid

- Mineralocorticoid excess
  - Hyperaldosteronism
  - Bartter syndrome
  - Cushing syndrome
- K loss
  - Urine Cl > 20 mmol/L

## Respiratory

### Causes of Acidosis

- Hypoventilation
  - CNS diseases
  - Muscle diseases
- Severe V/Q mismatch
  - Chronic lung diseases

### Causes of Alkalosis

- Brain lesion or diseases
- Centrally acting drugs or chemicals
  - Salicylate,
  - Endotoxin,
  - Progesterone
- Hypoxemia compensation
- Pneumonia, pulmonary edema, PTE
- Lack of fluid volume

- Liver failure
- Sepsis (+ met. acidosis)
- Psychiatric diseases

## Venous Blood Gas

Arterial blood gas sampling is an uncomfortable, painful, difficult and an invasive procedure for the patient. Furthermore, the success rate of the procedure may decrease due to movement of the patient or low arterial blood pressure. Therefore, the question “can venous blood gas be used instead of arterial blood gas?” has been raised, and many studies have been performed on this subject. Since venous blood gas is easy to sample from the peripheral veins or the central veins in patients with central venous catheters, it is a more comfortable and an easy procedure for some patients and the physicians.

In many studies, a very good correlation has been shown between venous blood gas and the arterial blood gas. To evaluate the acid-base disorders and

ventilation, comments can be made easily by checking the PvCO<sub>2</sub>, pH and HCO<sub>3</sub> levels. In addition, SvO<sub>2</sub> levels in patients with central venous catheters are very important indicators in evaluating patients in shock. However, it is not a useful method to evaluate oxygenation. To overcome this problem, it would be a sensible approach to measure the saturation with pulse oximetry simultaneously.

Arterial blood gas is a more reliable and accurate method for assessing the oxygenation. Arterial and venous blood gases provide similar and very close measurements in terms of PCO<sub>2</sub>, HCO<sub>3</sub>, and pH levels.

**Table 17.1** The comparison of arterial, peripheral vein and central blood gases

	PERIPHERAL VENOUS BLOOD GAS	CENTRAL VENOUS BLOOD GAS
PCO <sub>2</sub>	3 to 8 mmHg higher than the arterial pH	4 to 5 mmHg higher than the arterial pH
pH	0.02 to 0.04 pH units lower than the arterial pH	0.03 to 0.05 pH units lower than the arterial pH
HCO <sub>3</sub>	1 to 2 mEq/L higher than the arterial pH	little or no increase in HCO <sub>3</sub>

**References and Further Reading**, click [here](#)



# Cerebrospinal fluid analysis

by Arwa Alburaiki and Rouda Salem Alnuaimi

## Introduction

CSF is a colorless fluid that is present within the subarachnoid space, central canal of the spinal cord and the brain ventricles. It is produced at a rate of 500 ml per day, by the choroid plexus epithelial cells that are found in the brain ventricles [lateral, third and fourth ventricles], and reabsorbed back into circulation, by arachnoid granulation into dural venous sinuses. CSF is recycled about 2-3 times per 24 hr.

CSF circulates from the choroid plexus in the lateral ventricle into the third ventricle through the foramen of Monro, from the third ventricle into the fourth ventricle through the cerebral aqueduct of Sylvius. Then, it travels from the fourth ventricle into subarachnoid space through the

foramen of Luschka and foramen Magendie, where it will be reabsorbed again by the Arachnoid villi back to the venous drainage system of the brain.

## Normal CSF Composition

Color: Clear

WBC

- < 5 cells/mm<sup>3</sup> with less than 3PMN/mm<sup>3</sup> in adults
- <20 cells/mm<sup>3</sup> with < 1 PMN cells/mm<sup>3</sup> in neonate

RBCs: < 10 cells/mm<sup>3</sup>

Glucose: 45-80 mg/dl [CSF: serum ratio is 0.6] .

Protein

- <45 mg/dl in adults

- <20 mg/dl in children

Normal LP opening pressure

- In adults: 60-200 mmH<sub>2</sub>O [6-20 cm H<sub>2</sub>O].
- In children who are < 8 years 10-100 mmH<sub>2</sub>O.
- In neonate: 30-60 mmH<sub>2</sub>O

**Image 17.1**



## CSF Analysis and Interpretation

### Opening pressure

- Low Opening pressure: CSF leakage, or dehydration.
- High Opening pressure: overproduction, infection, bleeding, tumor, false measurement [sitting position, Valsalva or crying].

### Color

- Purulent :bacterial /TB meningitis.
- Xanthochromia [yellow color] in case of SAH, Hyperbilirubinemia.

**Image 17.2** Xanthochromia



The sample on the left represents xanthochromia. Retrieved from <http://www.medfriendly.com/xanthochromia.html>

## Cytology

### High WBC count

- Viral meningitis [ predominant lymphocytes].
- Bacterial meningitis [ predominant PMNs ].
- Fungal infection
- Vasculitis
- Traumatic tap

### High RBC count

- Taumatic Tap
- SAH

## Biochemistry

### Glucose level

- Decreases in case of bacterial/TB meningitis or CNS tumor.

### Protein

- Increases in:
  - Bacteria/TB meningitis.

- Blood [traumatic Taps /SAH).
- Multiple sclerosis.
- Guillain-Barre syndrome.

**References and Further Reading,**  
click [here](#)

### Miscellaneous test

- India ink for Cryptococcus
- VDRL/RPR for neurosyphilis
- PCR for HSV or CMV

**Table 17.2** CSF Analysis

	NORMAL	BACTERIAL	VIRAL	SAH
Color	Clear	Purulent	Clear/purulent	Bloody or xanthochromic
Opening Pressure (cm H <sub>2</sub> O)	7-18	>20 cm H <sub>2</sub> O	Normal/high	High
WBC/mm <sup>3</sup>	0-5	25-10000+	10-500	Slightly high
Differentials	Lymphocytes	PMNs	Lymphocytes	WBC/RBC ratio same to serum
RBC/mm <sup>3</sup>	0-5	Normal	Normal	>500
Glucose mg/100ml	45-80	<20	Normal/low	Normal
Protein mg/100ml	15-50	50-10000	50-200	60-150

*The source is not provided by authors.*

# Urine Analysis

by Jan Zajc

## Indications

Urinalysis should be performed to evaluate the following

- Evaluation of renal & lower urinary tract abnormalities
- Assessment of some metabolic/ endocrine disorders
- Assessment of hydration status

## Urine Collection

- Early Morning sample – qualitative
- Random sample – routine
- 24hr sample – quantitative estimation of proteins, Vanillyl mandelic acid, 5-hydroxyindole acetic acid, metanephrines, hormones in urine, microalbumin
- Midstream sample – UTI

- Postprandial sample – DM
- Catheterise – infants, bedridden patients
- Suprapubic needle aspiration

## Urine Examination

### Macroscopic Examination

**Normal Volume** – 600 – 2000mls

- Polyuria – Diabetes Mellitus, Diabetes Insipidus, Polycystic Kidney, Chronic Renal Failure, Diuretics.
- Oliguria – Dehydration, diarrhea, Excessive sweating, Acute glomerulonephritis, Acute tubular necrosis, Complete urinary tract obstruction

### Color



- Clear or yellow pale – normal due to pigments called urochrome
- Milky – Purulent UTI, chyluria
- Orange/Red – Urobilinogen, Red Beetroot ingestion, Hemoglobinuria, Haematuria
- Brown/Black – alkaptonuria, melanin

### Odor

- Normal – aromatic due to the volatile fatty acids
- Ammonical – bacterial action(E. coli) Fruity- ketonuria, starvation
- Musty – Phenylketonuria
- Fishy – UTI with Proteus
- Rancid – Tyrosinemia

### Urinary pH

- Normal pH 4.6 – 8

- Reflects the ability of the kidney to maintain normal hydrogen ion concentration in plasma & ECF

#### • Acidic urine

- Ketosis-diabetes,
- starvation, fever,
- systemic acidosis,

- UTI by E.coli,

- acidification therapy and high protein diet

#### • Alkaline urine

- Strict vegetarian,
- Systemic alkalosis,
- UIT by pseudomonas or Proteus,
- alkalinization therapy,
- CRF

### Specific Gravity

- Normal range- 1.003 to 1.035

#### •High

- All causes of oliguria,
- Glycosuria,
- DM,
- Dehydration,
- nephrotic syndrome

#### • Low

- All causes of polyuria except glycosuria DI,
- pyelonephritis,
- glomerulonephritis

### Osmolality

- Normal – able to produce 500-850 mOsm/kg water
- Dehydrated with normal renal function – 800 – 1400mOsm/kg water
- Diuresis with normal renal function – 40 – 80 mOsm/kg water

## Chemical Examination

### Proteinuria

- Glomerular proteinuria, e.g., nephrotic syndrome
- Tubular proteinuria: e.g., acute n chronic pyelonephritis, heavy metal poisoning, TB kidney
- Overflow proteinuria: Bence Jones proteins(plasma cell dyscrasia), hemoglobin( intravascular hemolysis), myoglobin(skeletal muscle trauma)
- Hemodynamic proteinuria: seen in high fever, hypertension, heavy exercise, CCF etc.
- Post-renal proteinuria: caused by inflammatory or neoplastic conditions in renal pelvis, ureter, bladder, prostate or urethra.
- Microalbuminuria – Defined as urinary excretion of 30 to 300 mg/24 hrs of albumin in the urine

- A prognostic marker for kidney disease
- in diabetes mellitus (earliest sign of renal damage in DM)
- in hypertension (sign of end-organ damage)
- increasing microalbuminuria during the first 48 hours after admission predicts an elevated risk for acute respiratory failure, multiple organ failure, and overall mortality
- Bence Jones proteins – monoclonal immunoglobulin light chains (kappa or lambda) synthesized by neoplastic plasma cells, seen in multiple myeloma, macroglobulinemias, primary amyloidosis

### Sugars – Benedict's test and Reagent Strip test

- Glycosuria with hyperglycemia
  - diabetes,

- acromegaly,
- Cushing's disease,
- hyperthyroidism,
- drugs like corticosteroids
- Glycosuria without hyperglycemia
  - renal tubular dysfunction

### Ketones

- Acetone, Acetoacetic acid,  $\beta$ -hydroxybutyric acid
- Non-diabetic causes- high fever, starvation, severe vomiting/ diarrhea, Glycogen storage disease

### Bilirubin

- Liver diseases
- Injury,hepatitis
- Obstruction to biliary tract
- Urobilinogen
- hemolytic jaundice

- Early hepatitis
- hepatocellular jaundice

## Blood

- Prerenal
  - bleeding diathesis,
  - hemoglobinopathies,
  - malignant hypertension
- Renal
  - trauma,
  - calculi,
  - acute & chronic glomerulonephritis,
  - renal TB,
  - renal tumors
- Postrenal
  - severe UTI,
  - calculi,

- trauma,
- tumors of the urinary tract

## Microscopic Examination

The centrifuged sample of urine sediment is examined on a glass slide under high magnification after the supernatant is discarded

**Acellular casts** – Hyaline casts, Granular, Waxy, Fatty, Pigment casts and Crystal casts

- Hyaline casts – Seen in fever, strenuous exercise, damage to the glomerular capillary
- Granular casts – indicative of chronic renal disease
- Waxy casts – severe longstanding kidney disease (end-stage renal disease)
- Fatty casts – nephrotic syndrome, diabetic or lupus nephropathy, Acute tubular necrosis

•Pigment casts – include those produced endogenously, such as hemoglobin in hemolytic anemia, myoglobin in rhabdomyolysis, and bilirubin in liver disease.

**Cellular casts** – Red cell casts, White cell casts, and Epithelial cell cast

- Red Cell casts – The presence of red blood cells within the cast is always pathologic, and is strongly indicative of glomerular damage, usually associated with nephritic Syndrome
- White Cell casts – Indicative of inflammation or infection, pyelonephritis, acute allergic interstitial nephritis, nephrotic syndrome, or post-streptococcal acute glomerulonephritis
- Epithelial casts – seen in acute tubular necrosis and toxic ingestion, such as from mercury, diethylene glycol, or salicylate

**Other structure** – Bacteria, Microfilaria, Trichomonas Vaginalis, Schistosoma haematobium, Spermatozoa, Yeast.

# How to perform urinalysis

Patients should be instructed clearly – using clean-catch, midstream specimen method is as accurate as catheterization. Urine should be checked immediately or refrigerated, but never left at room temperatures. In the tables are normal values.

Urine analysis **video**.

**References and Further Reading**, click [here](#)

**Table 17.3** Normal Urine Charactheristics

CHARACTERISTICS	FINDINGS
Color	Pale to dark yellow
Clarity	Clear
pH	4.5-7.4
Glucose	Negative
Protein	Negative
Ketones	Negative
Blood	Negative
Bilirubin	Negative
Urobilinogen	0.2 – 1.0
Specific gravity	1.005 – 1.025
Nitrite	Negative
Leukocyte esterase	Negative



# Whole blood cell count – CBC

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by Kaja Cankar and Gregor Prosen

## Introduction

The whole blood cell count is one of the most commonly ordered tests in medicine. It is a routine hematological screening study, performed to evaluate the status and overall health of a patient.

Whole blood cell count includes total red blood cell count (RBC) with indices, hemoglobin (Hb), hematocrit (HCT), white blood cell count (WBC) with or without a differential and a platelet level.

## Indications

Simplified guidelines suggest that whole blood cell count is indicated for

- trauma patients with acute blood loss and GCS of 8 or below,

- patients with acute illnesses of non-traumatic origin,

- which includes patients in emergency room presenting with fever, chest pain, abdominal pain, gastrointestinal bleeding, constipation, severe diarrhea, vomiting, severe nosebleed, irritability and crying (infants), throat pain, hypertensive urgencies and emergencies, severe joint pain or low back pain, skin rash, scrotal pain, seizures, syncope, vaginal bleeding, weakness and patients with lightning injuries, patients after near-drowning and patients with terrestrial venomous bites and stings.

## Red blood cell (RBC) count and indices

RBC count and indices can assist in determining whether our patient has anemia, polycythemia, and erythrocytosis.

Hemoglobin (Hb) reflects the amount of hemoglobin or oxygen-carrying potential available in the blood.

Hematocrit (Hct) indicates the proportion of whole blood that is occupied with red cell mass.

Special measurements of red cells, called indices, include:

- MCV Mean corpuscular volume or average size of the red cell.
- MCH Mean corpuscular hemoglobin or average hemoglobin content.
- MCHC Mean corpuscular hemoglobin concentration or average hemoglobin concentration.

- RDC Red cell distribution width measures the range of cell size.

## White blood cell count

WBC count includes differential with detail information about neutrophils, lymphocytes, monocytes, eosinophils, and basophils.

## Platelets

Platelet level is the number of platelets or thrombocytes in a given volume of whole blood. Both increased and decreased levels can point to abnormal conditions of excess clotting or bleeding.

## Variations In The Test Results

In a hospital setting, it is important to avoid taking blood from the same side as an infusion in order to avoid hemodilution. It should be taken into consideration that some samples that were difficult to obtain, e.g., lengthy venipuncture using a narrow gauge needle, such as a small butterfly, may

result in abnormalities due to cell lysis or clotting.

CBC in trauma patients may sometimes be misleading. It presents normal initial levels of hemoglobin which do not exclude a significant hemorrhage. Patient's hemoglobin value is not a real-time indicator of his or hers intravascular blood volume, and it takes quite some time (minutes-hours) before hemoglobin value reflects the degree of blood loss in trauma patients accurately. Following the trend of serial hemoglobin measurements, every 15 to 30 minutes can provide useful information regarding ongoing blood loss.

In trauma patients elevated white blood cell can often be found, but this occurrence is unlikely due to infection. WBC is elevated due to demargination of WBCs during the stress response.

## Interpretation of Test

### Increased WBC

- Infection (localized and generalized)

- Inflammation (i.e., vasculitis)
- Myeloproliferative disorder
- Tissue necrosis (burns)
- Myocardial infarction
- Physiological stress (e.g., exercise, pain, surgery, prolonged crying in infants)
- Medications (steroids)
- Vomiting
- Dysrhythmias
- Acute myocardial infarction (AMI),
- Pregnancy

The physician should look for a “left shift” which indicates the presence of immature forms in the peripheral circulation (bands). Usually, this represents an infectious state.

### Decreased WBC

- Infection (overwhelming sepsis or viral),

- Underlying hematopoietic disease (aplastic anemia, agranulocytosis)
- Immunosuppression,
- Medications (antibiotics, chemotherapeutic agents)

Patient presenting with neutropenia is at risk of infections from common and opportunistic organisms.

### Decreased HCT

- Blood loss
- Hemolysis
- Long-standing anemia
- Pregnancy

If suspecting acute loss, the physician should look for schistocytes on the peripheral blood smear. Long-standing anemia can be evaluated by the RBC indices. Administration of fluids in hypovolemic patients or trauma resuscitation will cause a decreased HCT.

### Increased HCT

- Hemoco concentrated states (dehydration, burns, diarrhea)
- High altitude,
- Exercise,
- Polycythemia Vera
- Chronic obstructive lung disease

### Decreased Hb

- Iron deficiency, vitamin deficiencies, e.g., vitamin B12
- Bleeding,
- Kidney disease
- Inflammatory disorders (rheumatoid arthritis or infections)
- Hemolysis (accelerated loss of red blood cells through destruction)
- Inherited hemoglobin defects (thalassemia or sickle cell anemia)
- Cirrhosis of the liver

- Bone marrow failure and cancers that affect the bone marrow

**Causes of increased Hb are similar to HCT.**

### Increased platelet count

- Myeloproliferative diseases
- Malignancy
- Infection
- Recent surgery (splenectomy)
- Chronic inflammation (i.e., irritable bowel syndrome)
- Trauma (massive hemorrhage, thrombus)
- Secondary to iron deficiency anemia or hemolytic anemia

In thrombocytosis, there is an excess of platelets (more than 1 million), but they are usually large and nonfunctioning.

### Decreased platelet count

- Infections (SBE, HIV, septicemia, mononucleosis)
- Drug-induced destruction (penicillin, heparin, sulfonamide, quinine)
- Idiopathic, thrombocytopenic purpura, thrombotic thrombocytopenic purpura, disseminated intravascular coagulation
- SLE
- Toxemia of pregnancy
- Renal insufficiency
- Bone marrow failure due to carcinoma, leukemia, lymphoma, or fibrosis
- Other: menses, poor nutritional states such as iron, folate, and vitamin B12 deficiencies.

### Hints And Pitfalls

Patients with serious infections may have completely normal, or even low WBC counts. Overreliance on normal WBC counts in the setting of acute infections may lead to misdiagnosis and delays in patient care.

Toxic granulations, Döhle Bodies, and cytoplasmic vacuolization are remnants of phagocytosis found in neutrophils. These are indicative of more serious bacterial infections.

Cutoff values of white blood cell (WBC) counts greater than 15,000/mm<sup>3</sup> suggest a higher likelihood of serious illness.

Acute hemorrhage will not be reflected in the Hgb or HCT early on.

Stress can cause the level of white blood cells to elevate, which can be misinterpreted as an infection.

In patients presenting with abdominal pain, an elevated WBC does not necessarily imply a serious disease.

### Pediatric, Geriatric, Pregnant Patient, And Other Considerations

- Geriatric patients will more than likely demonstrate normal to low WBC counts in sepsis.



- Elevated WBC can be found in prolonged crying in infants, pain, vomiting dysrhythmias, and pregnant patients.
- Pregnancy can lower the hematocrit by 10%.

CBC Tips and Notation – Simple Explanation of CNC Interpretation - [video](#).

**References and Further Reading**, click [here](#)

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## Chapter 18

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# Selected Imaging Modalities



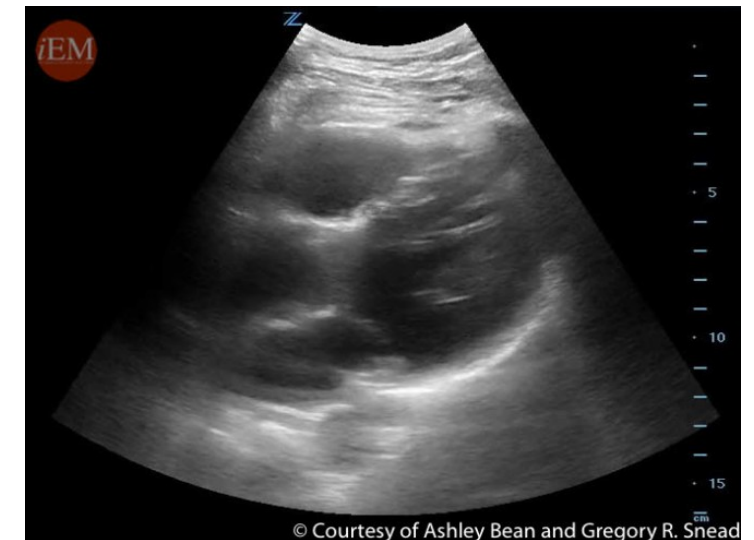
by Ashley Bean, Brian Hohertz and Gregory R. Snead

## Case Presentation

*A 40-year-old man involved in a car crash presents to your emergency department by ambulance. His vital signs are pulse 118 beats/minute, blood pressure 80/45 mmHg, respiratory rate 30 breaths/minute, oxygen saturation 98%, and temperature 37C. He is awake and oriented, complaining of epigastric abdominal pain and difficulty breathing.*

*The following images are obtained.*

**Image 18.1** Subcostal Cardiac



*Normal Subcostal Cardiac 4 chamber view*

**Image 18.2** Right Upper Quadrant



Audio is available [here](#)



**Image 18.4** Left Upper Quadrant



**Image 18.3** Suprapubic view



*After 1 Liter of normal saline, the patient remains hypotensive and is transferred*

*to the operating room where he undergoes a midline laparotomy. A spleen injury is identified intraoperatively.*

## Introduction

The objective of the extended focused assessment for sonography in trauma (eFAST) is to detect free fluid in the peritoneal, pleural and pericardial spaces, and also to detect free air in thoracic cavities. In the setting of trauma, we assume this fluid is blood; however, it can be urine or bowel contents as a result of organ rupture or it can be pre-existing ascites. An eFAST exam should take less than 5 minutes to complete.

In the peritoneal cavity, 200 ml of fluid can be detected via ultrasound in the ideal patient. In reality, however, the smallest detectable amount is usually around 500 ml. Hypotensive trauma patients with free abdominal fluid need urgent operative intervention. (Protocol 1) If there is not a surgeon who can repair

abdominal and cardiac injuries at your institution, transfer the patient to a facility with this capability. A stable patient with free intraabdominal fluid should undergo further diagnostic testing such as CT to ascertain the specific injury.

While peritoneal lavage has been traditionally utilized to evaluate for intraabdominal blood in the hypotensive trauma patient, the eFAST exam offers several advantages over peritoneal lavage. The eFAST exam is non-invasive, repeatable, rapid and sensitive for injuries requiring surgical intervention. It also does not interfere with computed tomography (CT) interpretation. Rozycki et al. reported that the FAST exam was shown to be 100% sensitive and 100% specific for hypotensive blunt abdominal trauma patients. Conversely, peritoneal lavage is invasive, can only be performed once, may require laboratory processing and has a high false positive rate. Peritoneal lavage may also confound interpretation of abdominal CT imaging.



Rapid detection of pericardial tamponade and cardiac injuries is of critical importance in the trauma patient. Fortunately, ultrasound is very sensitive for the detection of pericardial fluid. As little as 10 to 20 ml of fluid can be readily identified in the pericardium. In one of the original studies on the FAST exam, pericardial fluid had a sensitivity of 100% and a specificity over 99% for cardiac injury. Therefore, trauma patients with a pericardial effusion have a presumed cardiac injury requiring evaluation in the operating room. (Protocol 2)

Hypotensive trauma patients who do not have free fluid in their abdominal, pericardial, or plural spaces should be investigated for further injury. For instance, the patient may have spinal shock, a long bone fracture causing blood loss, or lost a significant amount of blood at the scene of the trauma. Other non-traumatic causes should also be considered such as myocardial infarction. Another possibility is that there is not yet a large enough amount of blood to be

detected by the eFAST exam in which case, the eFAST should be repeated.

## Procedure

The premise behind the eFAST exam is that free fluid accumulates in the dependent areas of the abdomen. An extended FAST exam involves several views. These are;

1. Subcostal or Parasternal Long Axis Cardiac
2. Right Upper Quadrant
3. Left Upper Quadrant
4. Suprapubic
5. Thorax for Hemothorax and Pneumothorax
6. IVC for volume status

Perform either a subcostal view or parasternal long axis view of the heart to look for pericardial effusion or tamponade. Views of the abdomen include the right and left upper quadrants as well as a suprapubic view. Image each

hemithorax for the presence of hemothorax or pneumothorax. Finally, the inferior vena cava is imaged to estimate the patient's volume status. While performing the exam, we ask four yes/no questions. These are;

1. Is there fluid in the peritoneal cavity?
2. Is there a pericardial effusion?
3. Is there fluid in the thorax?
4. Is there a pneumothorax?

## Emergency Indications

Indications for the eFAST include both blunt and penetrating traumatic injuries as well presentations of unexplained hypotension as part of an ultrasound shock protocol (i.e., RUSH exam) to rapidly diagnose the cause of low blood pressure.

## Contraindications

Contraindications to the eFAST are primarily situations in which performing the study would delay or interfere with

critical life-saving interventions including emergent surgical intervention.

## Equipment and Patient Preparation

Ultrasound equipment should be readily available in the emergency department setting. Machines should have low-frequency probes (2-5MHz) that may be either curvilinear or phased array in design (Image 5, 6). A high-frequency linear probe (5-10MHz) (Image 7) may also be used to aid diagnosis of pneumothorax in challenging cases and are preferred by some practitioners. Higher frequency curvilinear or phased array probes may be advantageous in pediatric cases. All machines should have the capability of image capture, preferably in digital format.

**Image 18.6** Curvilinear Transducer



**Image 18.5** Phased Array Transducer

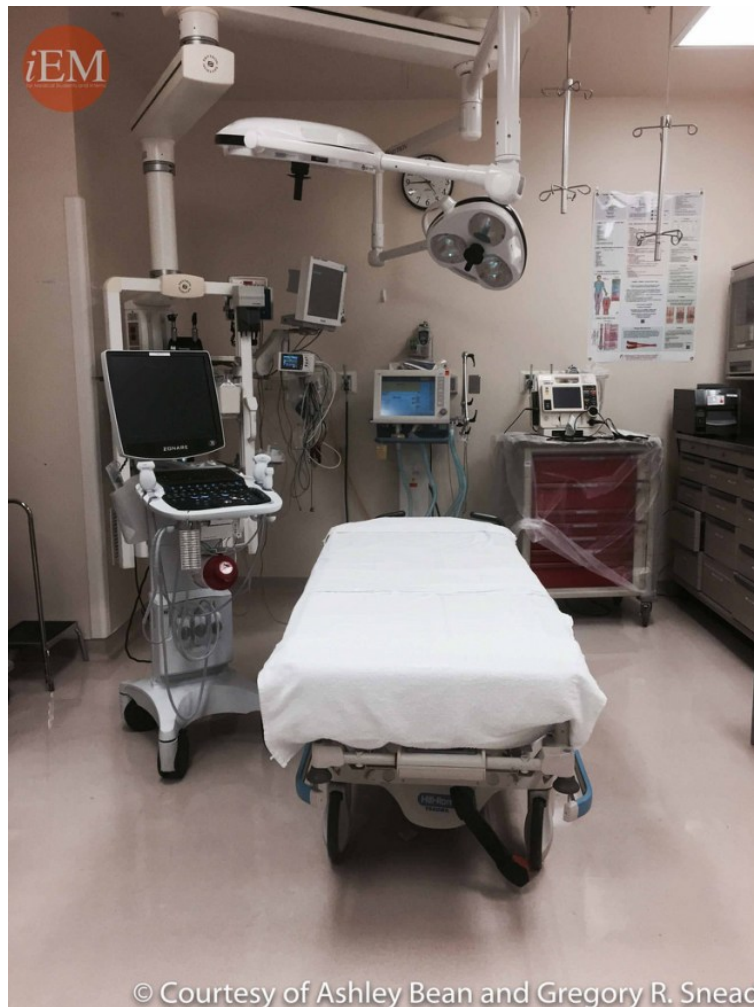


**Image 18.7** Linear Transducer



Prior to patient arrival, the ultrasound machine should be positioned to the left of the trauma bed and a patient identifier placed for the exam along with probe and study type selection (Image 8). Ultrasound gel should be stocked on the machine at all times. Following the primary survey, sufficient ultrasound gel should be applied to the epigastrium to facilitate all views. Please do not forget, ultrasound gel is cold. Therefore, awake patients should be informed. The exam should then be performed to include the recording of appropriate images and video clips.

**Image 18.8** Ultrasound and Patient Bed Position



## Transducer Orientation

Each transducer has a marker, which is oriented in the same direction as the probe marker indicator on the screen. For all of the views of the eFAST exam, except the parasternal long axis cardiac view, the probe marker should be pointed either towards the patient's head or right

side. When the transducer marker is point towards the patient's head (longitudinal orientation), the patient's head will be toward the left side of the screen, and their feet will be toward the right side of the screen. When the probe marker is pointed to the patient's right side, the patient's right will be toward the left side of the screen, and the patient's left will be toward the right side of the screen.

The orientation of the parasternal long axis view aligns with the axis of the heart rather than the external body. The probe marker is pointed toward the patient's right shoulder. (Image 18.9)

## Procedure Steps

Perform the eFAST exam immediately after the primary ATLS survey. Some authorities recommend applying e-FAST during the circulation phase of the primary survey, especially in hemodynamically unstable patients. It should be performed before the patient is rolled to minimize shifts independent fluid collections.

**Image 18.9** Transducer marker

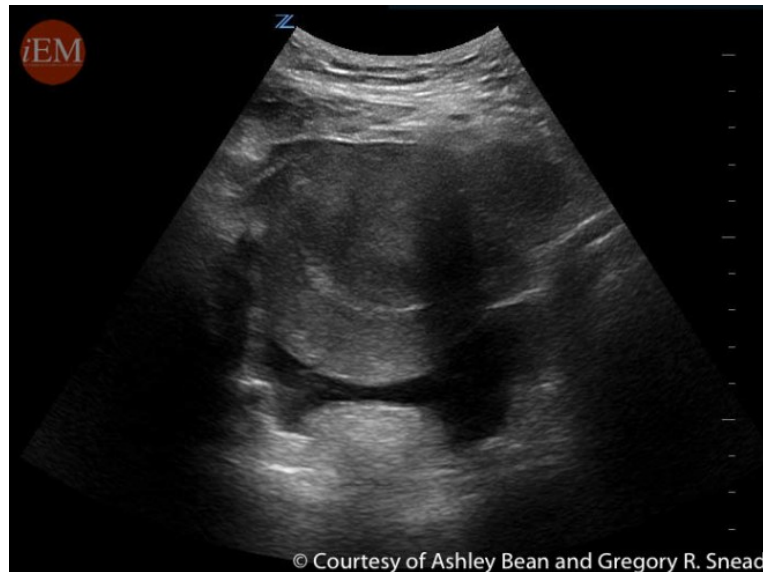


**Image 18.10** Longitudinal Orientation





**Image 18.11** Transverse Orientation (pelvis)



## 1. Imaging of the Heart for Pericardial Effusion and Tamponade

The first image obtained should be the heart. To obtain a view of the pericardial space, use either a subcostal or a parasternal long axis view. A low-frequency phased array or curvilinear probe should be used.

**Image 18.12** Subcostal Transducer Position



*To obtain this view, use the liver as the acoustic window.*

**Image 18.13** Normal Subcostal View



*The right ventricle is the closest cardiac chamber to the chest wall. The left ventricle and both atria are also visible. The bright white line is the pericardium.*

**Video:** Normal Subcostal Cardiac View. Again, the right ventricle is the closest cardiac chamber to the chest wall. The left ventricle and both atria are also visible. The bright white line is the pericardium. No anechoic fluid is visualized between the heart and the pericardium. There is a normal heart rate and good contractility.

**Video:** Large Pericardial Effusion. In this subcostal view, a pericardial effusion forms a black, anechoic rim around the heart.

**Video:** Pericardial Tamponade. The right ventricle, the closest chamber to the transducer, is collapsed, indicating that pressure from the pericardial fluid is inhibiting ventricular filling.

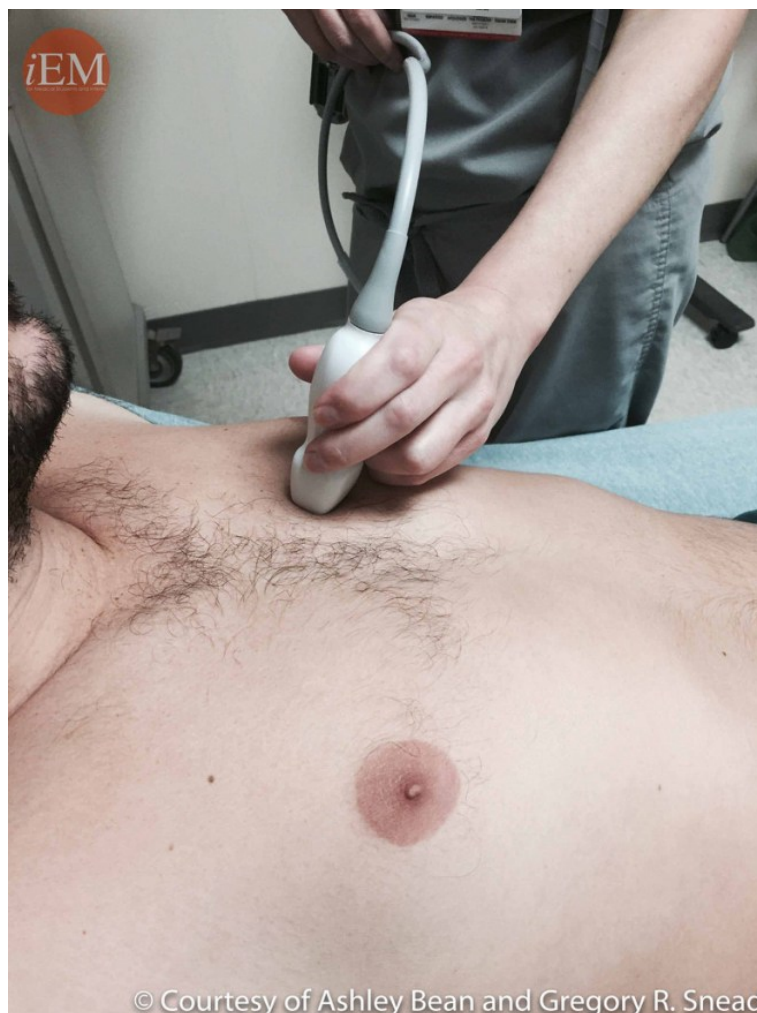
**Video:** Hypodynamic Heart. This patient has a hypodynamic heart with a low ejection fraction. To estimate ejection fraction, concentrate on the left ventricle. Decreased cardiac contraction may indicate that cardiogenic shock rather than hypovolemic shock is the cause of



the patient's hypotension. There is also a small pericardial effusion.

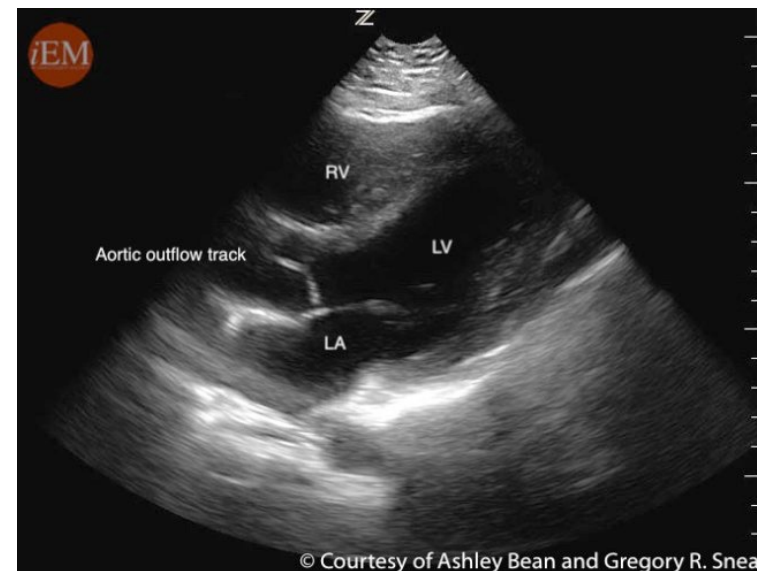
**Video:** Hyperdynamic Heart. The left ventricle has a high ejection fraction typical of someone with significant blood loss. The heart is attempting to circulate the remaining intravascular volume.

**Image 18.14** Parasternal Long Axis Transducer Position



**Video:** Normal Parasternal Long Axis View

**Image 18.15** Normal Parasternal Long Axis



*Normal Parasternal Long Axis Cardiac View. If an adequate subcostal view of the heart cannot be obtained, attempt the parasternal long axis view. A subcostal view might be difficult in some trauma patients if they have an abdominal injury, epigastric tenderness or abdominal distention. In this view, the right ventricle is still the closest cardiac chamber to the anterior chest wall. The left ventricle left atrium, and aortic outflow tract are also visible.*

**Video:** Pericardial Effusion. There is a large anechoic pericardial effusion

**Video:** Large Pericardial Effusion

**Video:** Hypodynamic Heart. This patient has congestive heart failure and a low ejection fraction. Look at the left ventricle to get a gross estimation of the heart's ejection fraction. Obtain an overall gestalt to label the ejection fraction as "normal," "high," "decreased," or "severely decreased."

**Video:** Hyperdynamic Heart. This patient has a hyperdynamic heart with an ejection fraction close to 100%.

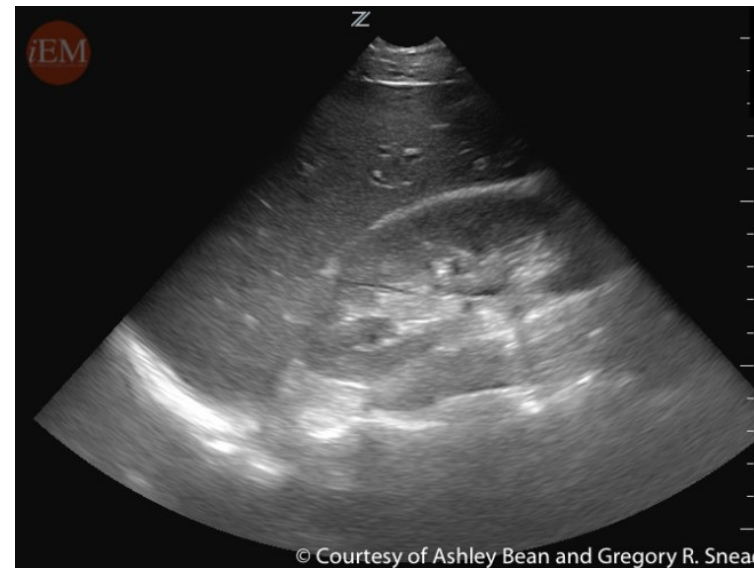
## 2. Imaging of the Abdomen for Free Intrapertitoneal Fluid

**Image 18.16** Right Upper Quadrant



*Right Upper Quadrant. Next, the abdomen is imaged for free fluid. The right upper quadrant is the abdominal view that is the most likely to be positive. The transducer is placed in the mid-axillary line with the probe marker pointed toward the patient's head. A low-frequency, curvilinear or a phased array probe should be used to obtain this view.*

**Image 18.17** Normal Right Upper Quadrant View



*Normal Right Upper Quadrant View. First, identify the kidney and the liver. The kidney is often the easiest structure to identify. It has a bright white center surrounded by the less echogenic cortex. The hepatorenal space, the interface between the kidney and liver, is a potential space that may contain free fluid. In this image, the patient's head is toward the left side of the screen, and the feet are toward the right side of the screen. The diaphragm is the bright white line just superior to the liver. It is important to visualize the tip of the liver as well as the inferior pole of the kidney to have an adequate assessment of the hepatorenal space.*

**Video:** Normal Right Upper Quadrant. Again, the probe marker is pointed toward the patient's head. This orientation corresponds to the probe marker indicator on the screen. There is



n of fluid collection between the kidney and the liver. As a patient breathes, the diaphragm lowers the position of the liver and kidney into a more inferior position.

**Image 18.18** Abnormal Right Upper Quadrant



*Abnormal Right Upper Quadrant. Note the small anechoic fluid collections between the liver and the kidney.*

**Video:** Abnormal Right Upper Quadrant. In this abnormal view of the right upper quadrant, there is a dark, anechoic stripe between the liver and the kidney. The liver is floating in a large amount of free fluid.

**Video:** Free Fluid. Free fluid typically has pointy edges. In contrast, fluid contained



within a lumen has rounded edges. The image shows free “pointy” fluid between the liver and the kidney.

**Video:** Luminal Fluid. The anechoic structure in this image is the gallbladder. Bile is an anechoic liquid, but since it is contained within the lumen the gallbladder, the edges appear rounded.

There are several other free fluid mimics. These are;

- Gallbladder
- Perinephric fat
- Stomach or duodenum
- Inferior vena cava

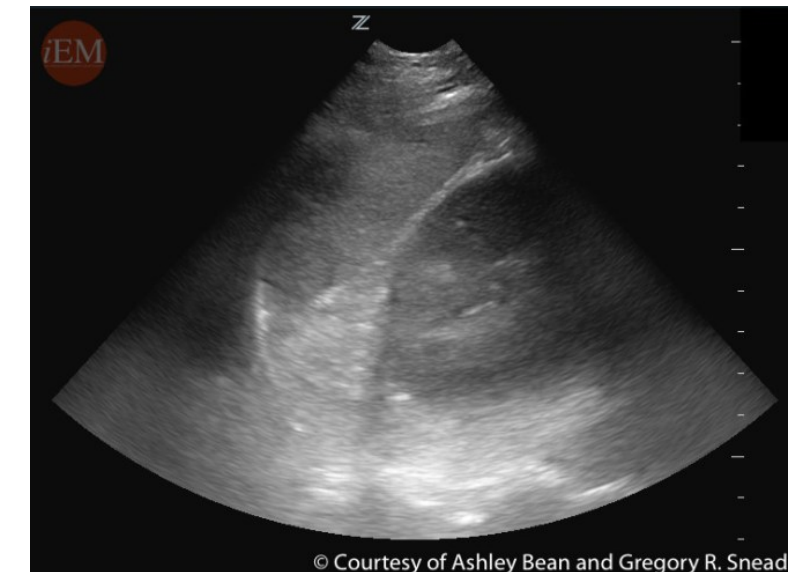
Perinephric fat can be mistaken for echogenic clot. However, this fat is usually symmetric, so compare with the opposite side. Fluid within the lumen of the stomach or duodenum should also have rounded edges. Fluid within the IVC should not only be rounded, but should show vascular flow with color Doppler.

**Image 18.19** Left Upper Quadrant



*Left Upper Quadrant. To obtain the left upper quadrant view, position the probe at the left posterior axillary line near ribs nine and ten. Again, the probe marker is pointed toward the patient's head. Since rib shadows may obscure your view, it is sometimes helpful to angle the probe obliquely in line with the intercostal space. Many novice sonographers do not position the probe posteriorly enough. The sonographer's hand should be parallel to and be resting on the bed. Since the spleen is smaller than the liver, the interface between the spleen and the kidney will be higher on the left side of the body. In awake, cooperative patients, asking the patient to take a deep breath may lower the spleen into the field of view.*

**Image 18.20** Normal Left Upper Quadrant



*Normal Left Upper Quadrant. This normal view of the left upper quadrant shows the spleen and the kidney. As with the right upper quadrant view, the patient's head is to the left of the screen, and the patient's feet are toward the right of the screen. The diaphragm is a bright white, hyperechoic curving line superior to the spleen. On the left side, examine the space between the kidney and the spleen for free fluid. However, it is more likely that fluid will accumulate around the dome of the spleen and, therefore, you must image the dome of the spleen.*

**Video:** Normal Left Upper Quadrant. In this video of a normal left upper quadrant, there is no collection of free fluid either between the spleen and the kidney or between the spleen and the diaphragm. There appears to be tissue with the same

echogenicity as the spleen superior to the diaphragm (the left of the screen). However, this is a mirror image artifact and, later in this chapter, we will discuss how the absence of this artifact can indicate fluid within the chest cavity.

**Video:** Abnormal Left Upper Quadrant. In this abnormal left upper quadrant view, there is fluid superior to the dome of the spleen. Imaging only the splenorenal interface would have missed this very abnormal finding. In the trauma patient with free intraperitoneal fluid, the spleen is the most commonly injured organ. However, the most likely location for fluid to accumulate is in the right upper quadrant. While this may seem counterintuitive, in a supine patient, the right upper quadrant is the most dependent area of the upper abdomen. So, in a patient with a positive intra-abdominal fast view, the location of the injury cannot be ascertained by the location of the fluid.

## Image 18.21 Pelvic Transvers View



*Pelvic View. The pelvic view is obtained by placing the transducer in a suprapubic position in either a transverse or longitudinal orientation. Since the bladder is the acoustic window, it is helpful to image the patient before the placement of a Foley catheter.*

Here the transducer is in a transverse orientation with the probe marker pointed toward the patient's right side (image 18.21). Angle the transducer so that its beam is pointed inferiorly in order to visualize the pelvic organs.

**Video:** Normal Pelvic View. This normal, transverse suprapubic view fans through the bladder. Look for free fluid lateral or inferior to the bladder.

**Video:** Normal Longitudinal Suprapubic View

**Video:** Abnormal Pelvic View. This video demonstrates free fluid adjacent to the bladder. The uterus is visualized floating within the fluid.

**Video:** Abnormal Pelvis View. Often the collection of free fluid is subtle as demonstrated by this retrovesicular collection.

## 3. Imaging of the Thorax for Hemothorax and Pneumothorax

To image the patient for the presence of a hemothorax, start with either the right or left upper quadrant view while angling the probe cephalad to image each hemithorax. The probe may need to be moved one rib space toward the patient's head. Focus on the diaphragm and look for the mirror image artifact of the liver or the spleen on the cranial side of the diaphragm. If the mirror image is absent, there is fluid within the pleural space.



## Image 18.22 Hemithorax Transducer Position



*Transducer Position. Make sure to angle the beam of the transducer into the chest cavity.*

## Image 18.23 Normal Hemithorax



*Normal Hemithorax. This image shows the kidney, liver, and diaphragm. However, as you transition to the chest cavity at the insertion of the diaphragm, air scatters the ultrasound beam, and you lose visualization of the spine. In addition, you can see a mirror image artifact.*

## Image 18.24 Hemothorax or Pleural Effusion



*Hemothorax or Pleural Effusion. In this image, there is a normal hepatorenal space. The diaphragm is visualized at its insertion. Rather than a mirror image, there is an anechoic fluid collection, and the thoracic vertebrae are visible. In addition, there is a "spine sign." Usually, the spine cannot be visualized within the thoracic cavity because air scatters the ultrasound beam. If you are able to visualize the spine, then there is a medium present (free fluid) which can transmit the sound wave.*

**Video:** Normal hemithorax. This video demonstrates a normal right upper quadrant and hemithorax with a mirror image artifact.

**Video:** Small Pleural Effusion or Hemothorax. In this video of the chest, there is an anechoic, pleural effusion rather than the mirror image. The spine is visualized in the chest cavity. The tip of the lung floats into the picture as the patient breaths.

**Video:** Free fluid within both the right thoracic and abdominal cavities. Fluid is visible both superior to and inferior to the diaphragm.

## Image 18.25 Transducer Position to Evaluate for Pneumothorax



*The extended FAST exam also images each hemithorax for pneumothorax.*

Lung sliding rules out pneumothorax in the segment of lung the transducer is imaging. Since air rises, the transducer should be placed at the most superior region of the chest. In the supine, trauma patient, this position is usually the third intercostal space. If the patient is sitting, the apices of the lungs should be imaged. A high-frequency probe should be placed in a longitudinal position with the indicator pointed toward the patient's head.

**Image 18.26** Normal Lung



The patient's head is to the right of the screen, and his feet are to the left. Look for a rib and a rib shadow as landmarks to help find the pleural line. The bright, white light line is the opposition of both the visceral and the parietal pleura and should shimmer, moving back and forth (a sliding motion) with respirations.

**Video:** Normal Lung. In this video of normal lung, identify the superior rib (left side of the screen) and the inferior rib (right side of the screen) with their corresponding rib shadows. The bright white line between the two is the plural line and can be seen sliding or shimmering.

**Image 18.27** Pneumothorax



The inferior rib and rib shadow are still visible, but only the visceral pleura is visualized. The parietal pleura covering the surface of the lung has dropped away from the chest wall. The two pleural layers no longer slide over each other.

**Video:** Pneumothorax. Recognize the inferior and superior ribs with their corresponding rib shadows and the bright light line is the parietal pleura. Note this line is not sliding with respirations.

**Video:** Pneumothorax. This video shows another example of a pneumothorax.

Absent lung sliding can be associated with pneumothorax; however, lack of lung sliding does not “rule in” a pneumothorax. Pneumothorax would be the most likely diagnosis; however, there are several conditions you should consider. Lack of sliding may be observed in patient who are not breathing, who have a mainstem intubation, or in cases where the pleura is adherent to the chest wall.

## 4. Vena Cava Imaging for Volume Assessment

The inferior vena cava (IVC) normally has respiratory variation. In a patient with normal volume status, the IVC will collapse 30-70 percent as the patient inhales. The IVC caliber of hypovolemic



patients will be smaller and collapse greater than 70%. Conversely, patients with fluid overload will have an enlarged IVC with minimal collapse.

**Image 18.28** Transducer Position for Volume Assessment



*Place the transducer on the abdomen with the probe marker pointed towards the patient's head. Visualize the vena cava about 3 cm proximal to the cavoatrial junction.*

**Video:** Euvolemia. This video demonstrates a normal inferior vena cava in a patient who is euvolemic. There is respiratory variation in the vena cava, but the collapse is not greater than 30%.

**Video:** Hypovolemia. The walls of the vena cava completely collapse with respiration in this hypovolemic patient.

IVC collapse estimates the patient's volume status. It does not predict the patient's response to hydration.

**Video:** Volume Overload. There is a large vena cava with minimal change with respiration.

## Hints and Pitfalls

- If initial eFAST exam is negative, but you have continued concern, repeat the exam.
- Repeat the eFAST exam if there is a change in the clinical status.
- Perinephric fat may be mistaken for clot; however, it is usually symmetrical. Examine the opposite side and compare.
- Placing the patient in reverse Trendelenburg may help visualize free fluid.

• Always remember that free fluid may not be blood – consider ascites, bladder rupture, and bowel rupture as causes of free intraperitoneal fluid.

- Since the bladder is your acoustic window, the pelvic view should be imaged prior to insertion of catheter.
- A normal echo does not definitively rule out major pericardial injury.
- An epicardial fat pad may easily be misinterpreted as “clot.”
- Hemothorax may be confused with pericardial effusion.

## Post Procedure Care and Recommendations

Post-procedure care consists of clear communication of the results to the trauma team (positive, negative, or indeterminate for abdomen, thorax, and pericardium). Limited views should be discussed and scanning repeated by the most experienced sonographer. Adjunctive maneuvers to improve

visualization such as repositioning the patient, or filling the bladder via foley catheter to obtain a better view of the pelvis should be considered. Clean ultrasound gel off the patient to help maintain body temperature. Clean and decontaminate the ultrasound machine based on your institutionally approved process by removing surface gel and using an appropriate surface wipe or process. Complete any additional documentation of the images along with a note describing the procedure and findings for inclusion into the medical record.

## Cautionary Note

Complications of the eFAST are typically a result of incorrect performance or interpretation of results leading to false positive or false negative results. Difficult or limited exams should be discussed or repeated by the most experienced sonographer on the resuscitation team. Team leadership should also interrupt or delay the eFAST for critical interventions in the care of the patient. And, please keep in your mind, e-FAST should not delay the definitive treatment of trauma patient.

## Pediatric, Geriatric and Pregnant Patient Considerations

In pediatric patient, the eFAST is highly specific but has insufficient sensitivity to exclude intra-abdominal injury. Though no change in test performance characteristics have been reported related to probe choice, consider using a higher frequency probe in smaller patients.

Pregnant patients present several challenges in clinical assessment and use of the eFAST exam. Clinical instability may require placing the patient in the lateral position to maximize blood flow to the uterus and require repositioning to complete the exam. Uterine enlargement can limit the view of the bladder but also result in displacement of bowel loops making pelvic views variable and occasionally dependent on fetal positioning. Late gestation is accompanied by other changes in addition to uterine enlargement including diaphragmatic elevation that may require repositioning the probe to achieve adequate views. Test performance has been reported to mirror those in non-pregnant patients in spite of these challenges. Another important requirement is rapid fetal assessment in trauma presentations. Rapidly determining the fetal heart rate should be determined on arrival and will likely precede initiation of continuous fetal monitoring by the obstetric team. Fetal reassessment should be regularly performed until continuous monitoring is available. Remember that ultrasound cannot exclude placental abruption – even in seemingly low force scenarios. Obstetric consultation and prolonged fetal monitoring is advised in all trauma cases involving a fetus of potentially viable gestational age.

**References and Further Reading**, click [here](#)



# PoCUS – RUSH Protocol

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by Rasha Bhumaid

## Why use POCUS in undifferentiated hypotension?

Hypotension is a high-risk sign which is associated with increased morbidity and mortality rate. The differential diagnosis for hypotension is broad and the treatment depends on the underlying etiology. In most cases of hypotension, patients present with limited history and physical examination may be inaccurate making the management of the condition a great challenge for emergency physicians.

The use of POCUS in undifferentiated hypotension has been shown to help correctly and rapidly identify the etiology and therefore initiate the appropriate management. Since 2001, there are many protocols published describing a systematic approach to the use of POCUS in undifferentiated hypotension. Table 18.1 summarizes the components of these protocols. In this chapter, we will review the Rapid Ultrasound in Shock (RUSH) protocol.

Equipment: Ultrasound machine with a linear probe (10-5 MHz) and phased array (5-1 MHz) or curved array (5-2 MHz) probe.

## The RUSH protocol

The RUSH protocol (Rapid Ultrasound in Shock and Hypotension) uses the analogy of plumping system to categorize the causes of shock into three categories: The pump, the tank, and the pipes. The categories are assessed using specific and straightforward questions described below.



Audio is available [here](#)

**Table 18.1** Protocols for undifferentiated shock

<div> <div>iEM</div> <div>Table 1: Summary of components of the protocols used for undifferentiated shock</div> </div>						
Protocol	UHP	Jones	RUSH	ACES	RUSH	FAST Reliable
Year Published	2001	2004	2008	2009	2010	2012
Abdominal Ultrasound	X	X	X	X	X	X
RV dilation		X	X	X	X	X
Pericardial effusion	X	X	X	X	X	X
LV function		X	X	X	X	X
IVC evaluation		X	X	X	X	X
Aorta evaluation	X	X	X	X	X	X
DVT					X	X
Pneumothorax			X		X	X
Pleural effusion			X	X	X	X
Pulmonary edema					X	
Ectopic pregnancy						X

Prepared by Rasha Buhumaid

## Step 1 – Pump: Cardiac Evaluation

The basic echocardiography views summarized in this tutorial ([Video](#)).

This step is used to evaluate the pump (the heart) for the following:

### A. Determine the presence of pericardial effusion

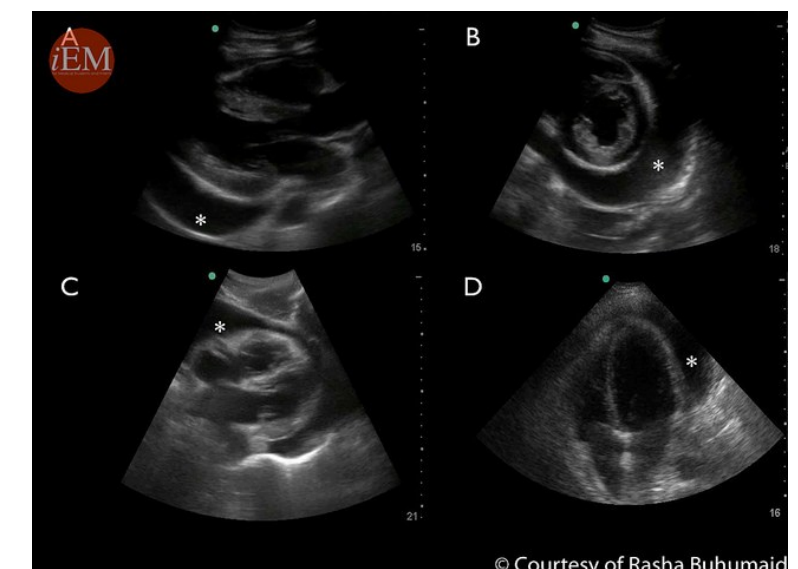
Pericardial effusion will lead to hypotension when the fluid surrounding the pericardium causes obstructive physiology preventing the venous return to the heart. This is known as cardiac tamponade. Pericardial effusion and tamponade can easily be identified using POCUS.

Technique: any of the above described basic echocardiography views can be

used to assess for pericardial effusion.

Interpretation: Pericardial effusion will appear as anechoic (black) fluid surrounding the heart (Image 18.29). The ultrasound finding of cardiac tamponade includes circumferential pericardial effusion with hyperdynamic heart swinging in the pericardial sac ([Video](#)). Right atrial wall diastolic collapse and right ventricular early diastolic collapse is also known as scalloping ([Video](#)).

**Image 18.29** Pericardial effusion



Pericardial effusion marked by \* in parasternal long view (A), parasternal short view (B), subxiphoid view (C) and apical 4 chamber view (D)

**Video:** Circumferential pericardial effusion with hyperdynamic left ventricle and swinging of the heart in the pericardial sac in a parasternal long view (A) and parasternal short view (B)

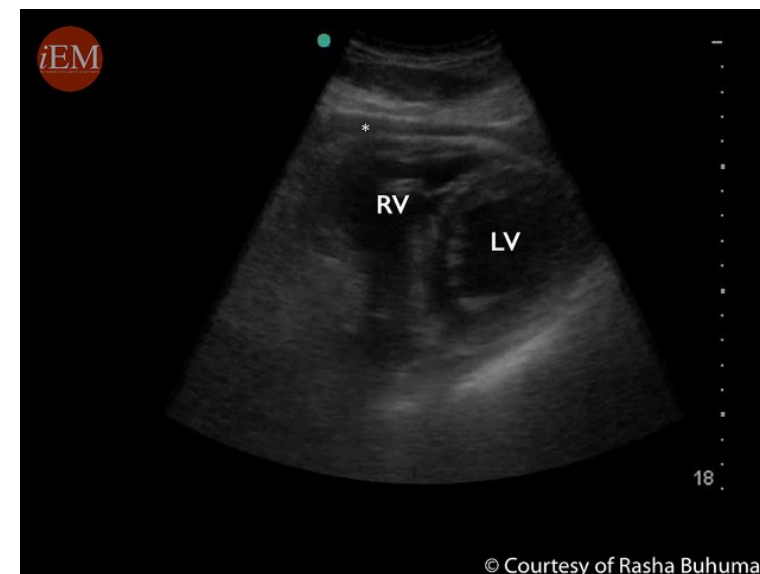
**Video:** Subxiphoid view demonstrating pericardial effusion with diastolic collapse of the Right Atrial (RA) wall

**Pitfalls:** Two important conditions can be confused with pericardial effusion.

Pericardial fat pad (Image 18.30) which is not usually anechoic (black) but rather has some echogenicity (gray) and it is usually confined to the anterior wall above the right ventricle.

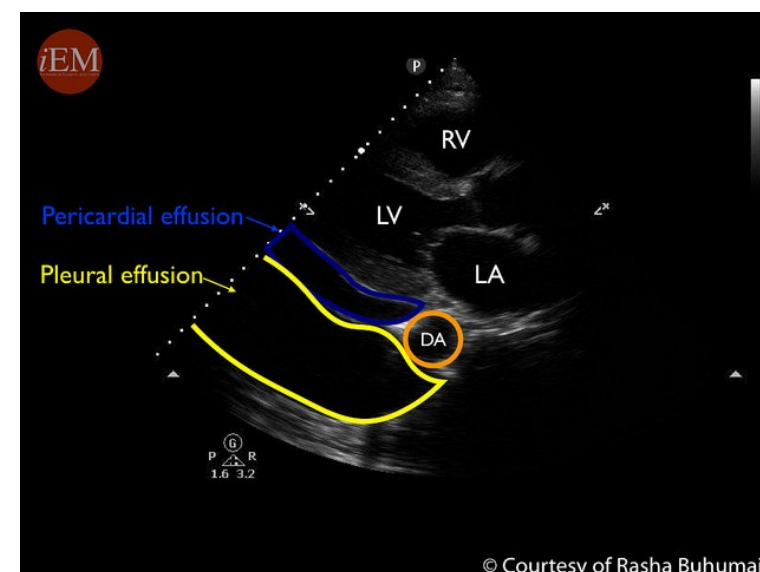
Pleural effusion can be confused with pericardial effusion in a parasternal long view. This can be differentiated using the descending aorta as a landmark in parasternal long view. A pericardial effusion will track above the descending aorta while pleural effusion will track below the descending aorta (Image 18.31).

**Image 18.30** Pericardial fat pad



*Fat pad marked by \* above the Right Ventricle (RV) wall in a parasternal short view*

**Image 18.31** pericardial effusion



*Parasternal long view demonstrating pericardial effusion tracking above the Descending Aorta (DA) and pleural effusion tracking below the DA*

## B. Assess Left Ventricular (LV) contractility

In the setting of hypotension, severely decreased LV contractility can help identify possible cardiogenic shock while hyperdynamic LV could result from severe hypovolemia, acute blood loss or early septic shock.

**Technique:** Any of the basic echocardiography views can be used to assess LV contractility.

**Interpretation:** The contractility of the LV can be evaluated by visual assessment of the difference in the LV volume between the end of systole and diastole. In normal LV contractility, there is a significant change in the volume of the LV between systole and diastole (**Video**). However, in a poorly contracting LV, there is a small change in the size of the LV volume during the cardiac cycle (**Video**). Using this method, the LV contractility can be broadly categorized into 4 categories; normal, mild to moderately decreased, severely decreased and hyperdynamic LV.

Hyperdynamic LV is when the volume changes between systole and diastole more than 70% or the walls of the LV touch during systole (**Video**). Studies have shown that qualitative visual assessment correlates well with the quantitative techniques used by cardiologists.

**Video:** Normal Left Ventricular (LV) contractility in parasternal long view (A) and parasternal short view (B)

**Video:** Severely decreased LV contractility in parasternal long view (A) and parasternal short view (B)

**Video:** Hyperdynamic LV contractility in parasternal long view

Pitfalls: In some cases of cardiogenic shock, LV contractility will be preserved, for example, acute severe valvular regurgitation and acute right ventricular infarction.

### C. Assess for Right Ventricle (RV) strain

In the right clinical setting, hypotension with signs of RV strain could be a sign of massive pulmonary embolism causing obstructive shock.

Technique: using parasternal short view and apical 4 chamber view can identify signs of RV strain.

Interpretation: the signs of RV strain include 1) RV enlargement: best assessed in apical 4 chamber view. Normally, the RV is smaller than the LV with a normal RV:LV ratio of 0.6:1. If RV/LV ratio  $> 0.9$  it suggests RV enlargement (**Video**). 2) The D sign: In a parasternal short view, bowing of the interventricular septum towards the LV indicate increase pressure in the RV (**Video**).

**Video:** Apical 4 chamber views with normal Right ventricle (RV) size (A) and enlarged RV (B)

**Video:** Parasternal short views with normal interventricular septum (A) and D sign: bowing of the interventricular septum towards the LV (B)

Pitfalls:

- The apical 4 chamber view can be a challenging view to obtain due to technical difficulty the view might be foreshortened (the apex appears rounded rather than bullet shape), this will lead to overestimation of the RV size.
- An important pitfall to consider when evaluating for signs of RV strain is that RV dilation is not specific for massive pulmonary embolism. Any condition that increases the pressure of the right heart will lead to dilated RV including chronic COPD and pulmonary hypertension. However, the RV wall is likely to be thick in chronic conditions.
- It is important to note that normal RV on echocardiography does not rule out pulmonary embolism.

## Step 2 – Tank: Volume Status Evaluation



## A. Tank fullness: Inferior Vena Cava (IVC) evaluation

In spontaneously breathing patient, during inspiration, the negative intra-thoracic pressure will increase venous return to the heart leading to IVC collapse which is reversed during expiration. This physiology is reversed in ventilated patients. During insufflation, the intra-thoracic pressure increases, decreasing the venous return and the IVC expands, this is reversed during expiration. Studies have shown that the degree of IVC size change during the respiratory cycle can be used to predict volume responsiveness.

Technique: using a low frequency probe in a longitudinal plane in the subxiphoid area the IVC is identified as a vessel draining into the right atrium (**Video**). The IVC can be assessed using 2 methods: qualitative and quantitative. The qualitative method is by visual assessment of the size and collapsibility with respiratory cycle (**Video**). The quantitative method is by calculating the

percentage of IVC collapsibility with respiratory variation also known as the Caval Index (CI). Using M mode, the IVC size measured 2 cm from the right atrial junction during inspiration and expiration. The CI (%) = (IVC expiratory diameter – IVC inspiratory diameter) / IVC expiratory diameter × 100 (Image 18.32).

**Video:** Tutorial on ultrasound of the Inferior Vena Cava

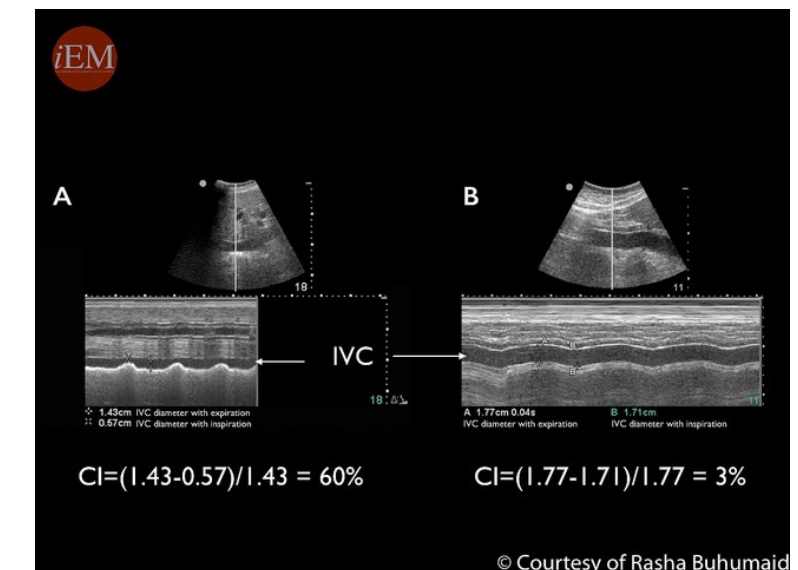
**Video:** A: small and collapsible IVC with respiration. B: large and non-collapsible IVC with respiration

Interpretation: In spontaneously breathing patients, collapsible IVC or caval index more than 40% is associated with volume responsiveness. While dilated non-collapsible IVC does not rule out volume responsiveness.

Pitfalls:

- The IVC can be mistaken for the abdominal aorta. This can be avoided by visualizing the IVC drain into the

**Image 18.32** M mode evaluation of the IVC



M mode evaluation of the IVC: (A) shows a small and collapsible IVC with respiration and Caval Index= 60%. (B) shows a large IVC with minimal respiratory variation and Caval Index = 3%

right atrium while the abdominal aorta passes behind the heart.

- Interpreting the findings of IVC assessment alone can be misleading as there are numerous causes of dilated non-collapsible IVC including tension pneumothorax, massive pulmonary embolism and pericardial tamponade.

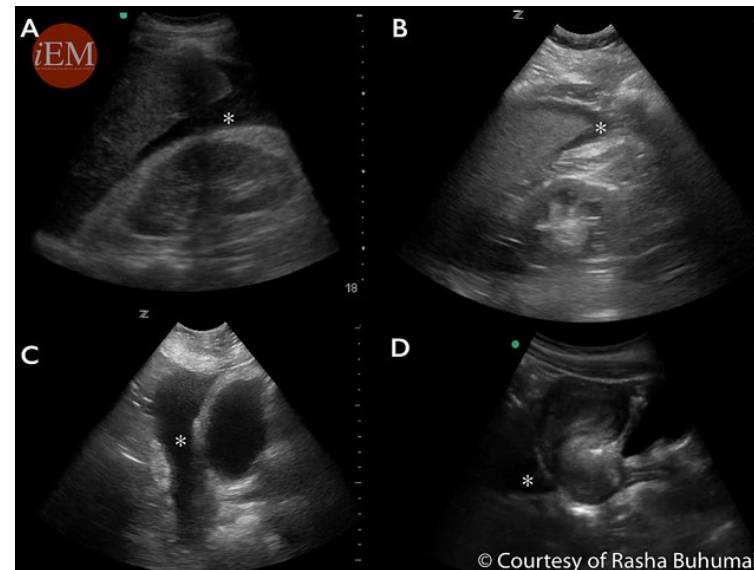
## B. Tank Leakiness: FAST Exam

In atraumatic hypotension, intrabdominal free fluid could be due to ruptured abdominal aortic aneurysm, ruptured ectopic pregnancy or ruptured hemorrhagic ovarian cyst.

Technique: the scanning technique for Intraperitoneal free fluid is described in details in the **eFAST chapter**.

Interpretation: Intraperitoneal free fluid is identified as anechoic (black) fluid collection in any of the following areas. In the right upper quadrant view, fluids will accumulate in the hepatorenal space also known as Morrison's pouch. In the left upper quadrant view, fluids will accumulate in the subdiaphragmatic space that can extend into the splenorenal space. In the pelvic view, fluid will accumulate in rectouterine space also known as the pouch of Douglas in females and rectovesical space in males. (Image 18.33)

**Image 18.33** FAST exam demonstrating free fluid



FAST exam demonstrating free fluid marked by (\*) in the hepatorenal space (A), subdiaphragmatic and splenorenal space (B), rectovesical space (C) and pouch of Douglas (D)

### Pitfalls:

Free fluid in the peritoneal cavity can be urine or previous ascites. Ultrasound cannot differentiate these fluids. However, in a patient with shock and hypotension, this free fluid is considered as blood until proven otherwise.

## C. Tank Overload: Assessment of pleural effusion and pulmonary edema

In the setting of hypotension, decreased LV contractility, dilated non-collapsible IVC, signs of pulmonary edema and bilateral pleural effusion suggests cardiogenic shock.

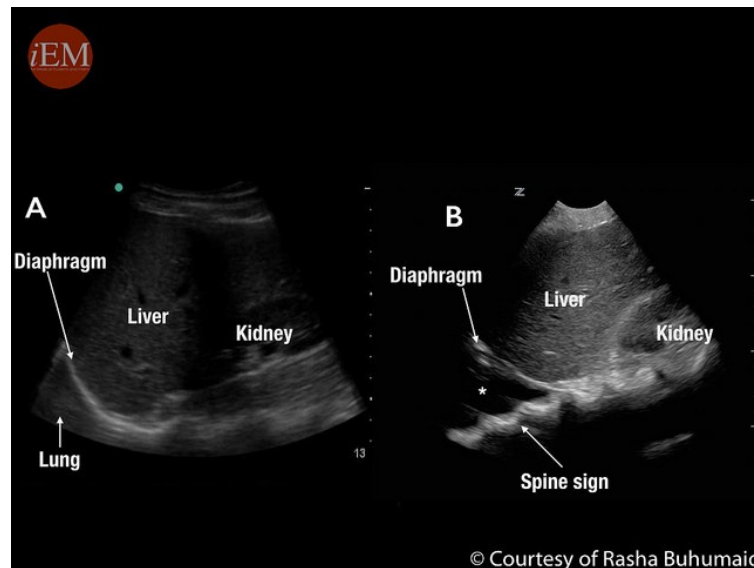
Technique: Using a low frequency probe, assess for pleural effusion by obtaining the same views used for the FAST exam. Start with the coronal views of the right upper quadrant and left upper quadrant then move the probe cephalad (towards the head) to visualize the pleural cavity better; space above the diaphragm (**Video**). To assess for pulmonary edema, use a low frequency probe to obtain a longitudinal lung view of the anterior chest wall, locate two ribs (hyperechoic area with posterior shadowing) and identify the pleural line (hyperechoic line) between the rib shadows (**Video**).

**Video:** Tutorial on lung ultrasound for pleural effusion

**Video:** Tutorial on lung ultrasound for pulmonary edema

Interpretation: Pleural effusion is confirmed if a black/anechoic fluid is identified above the diaphragm or by visualizing the thoracic vertebra body (hyperechoic-white line with posterior shadowing) extending above the diaphragm also known as positive spine sign (Image 18.34).

**Image 18.34**



(A) Right upper quadrant view with normal pleural space. (B) Right upper quadrant view with evidence of pleural effusion (\*) and positive spine sign

In normally aerated alveoli, the pleural line will produce reverberation artifact known as A-lines. However, in case of fluid-filled

alveoli, lung ultrasound will produce B lines: hyperechoic-white vertical lines extending from the pleural line into the far field. In case of pulmonary edema, B lines will be bilateral and in all lung zones (**Video**).

**Video:** Lung ultrasound (A) A-lines in the normal aerated lung. (B) B lines extended from the pleural line to the far field indicating fluid-filled alveoli

Pitfalls: B-lines are not specific for pulmonary edema. Any pathology that will fill the alveoli with fluids including ARDS and bilateral pneumonia will produce bilateral B-lines on lung ultrasound.

## D. Tank Compromise: Assessment for pneumothorax

Tension pneumothorax is another cause of obstructive shock.

Technique: using a high frequency (linear) probe, obtain a longitudinal view of the lung between 3rd and 4th intercostal space, mid-clavicular line (**Video**).

**Video:** Tutorial on lung ultrasound for pneumothorax

Interpretation: Assess the pleural line for sliding with respiration either by visual evaluation or using M-Mode. The presence of lung sliding, seen as seashore sign on M mode rules out pneumothorax with 100% negative predictive value. In the absence of lung sliding also seen as barcode sign on M mode (**Video**) continue following the pleural line inferiorly and laterally to identify the boundary of pneumothorax known as lung point. A lung point on ultrasound will appear as a boundary between the absence of pleural sliding and normal pleural sliding (**Video**). The lung point is very specific for pneumothorax.

**Video:** (A) Lung ultrasound with normal pleural sliding. (B) Lung ultrasound with absence of pleural sliding. (C) M mode demonstrating seashore sign indicating normal pleural sliding. (D) M mode

demonstrating barcode sign indicating absence of lung sliding

**Video:** Lung point which is the boundary between absence of lung sliding and normal lung sliding

Pitfalls: The absence of lung sliding could be from numerous causes other than pneumothorax including: pleurodesis, pleural bleb, poor respiratory effort and mainstem intubation.

## Step 3 – Pipes: Vascular system Evaluation

### A. Assessment for Abdominal Aortic Aneurysm (AAA)

A ruptured aortic aneurysm is a cause of non-traumatic hemorrhagic shock.

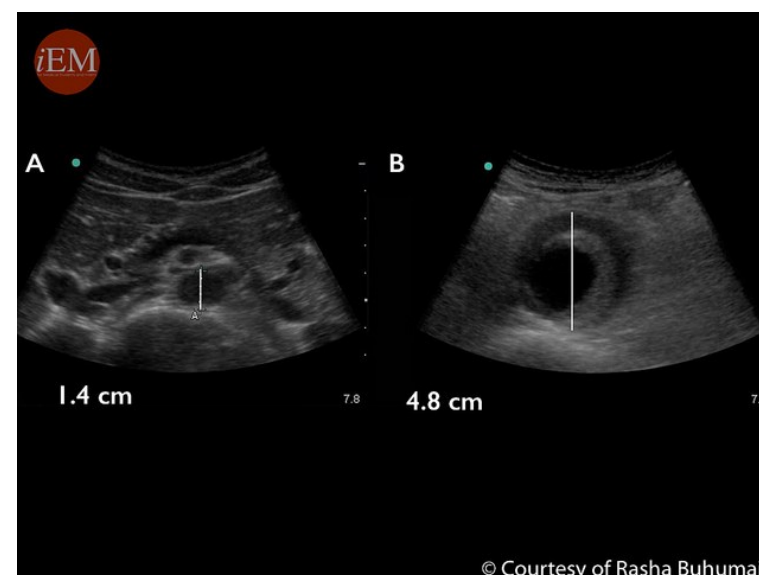
Technique: Using a low frequency probe. The aorta is scanned in a transverse view starting in the subxiphoid area. The landmark used to identify the aorta is the vertebral body (hyperechoic-white structure with posterior shadowing). The aorta is located anterior and to the left of

the vertebral body. The aorta is traced caudad (toward the feet) until it bifurcates into the common iliac arteries at the level of the umbilicus (**Video**).

**Video:** Tutorial on ultrasound for abdominal aortic aneurysm

Interpretation: The abdominal aorta is measured from outer wall to outer wall at the proximal, middle and distal aorta. Normal abdominal aorta measures less than 3 cm. AAA is defined as abdominal aorta measuring greater than 3 cm (Image

**Image 18.35** Aorta evaluation



(A) Normal abdominal aorta. (B) Abdominal aortic aneurysm measuring 4.8 cm with intramural thrombus

18.35) and the risk of rupture increases when it is greater than 5 cm.

Pitfalls: The majority of ruptured AAA are retroperitoneal therefore when performing the FAST exam, no intraperitoneal free fluid will be identified. Always make sure that you are measuring the aorta from the outer wall to outer wall.

### B. Assessment for clogged pipes: Deep Venous Thrombosis (DVT)

When considering pulmonary embolism as a cause of obstructive shock, obtaining adequate echocardiography views to assess for RV strain can be challenging. In this situation, evaluating the extremities for DVT can be used as a surrogate marker for possible pulmonary embolism as literature shows that majority of pulmonary embolism originate from DVT.

Technique: using a low-frequency probe the 2 zone graded compression technique is used to identify DVT.



1) Assess the common femoral vein zone: place the probe in a transverse plane just below the inguinal ligament, identify the common femoral vein and greater saphenous vein. Trace the common femoral vein distally until it divides into the superficial and deep femoral vein.

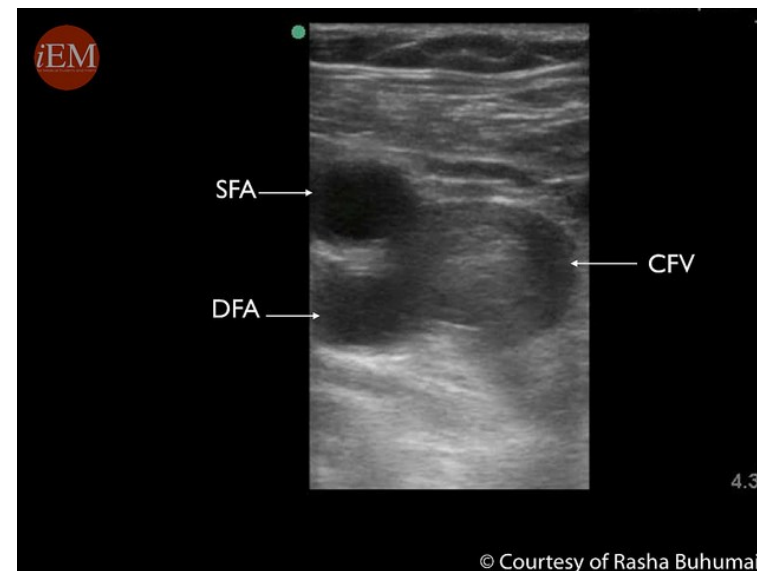
2) Assess the popliteal zone: place the probe in a transverse plane in the popliteal fossa, identify the popliteal vein which is located on top of the popliteal artery. Trace the popliteal vein until it trifurcates distally. Apply graded compression on all the veins identified to ensure complete collapsibility of the veins (**Video**).

**Video:** Tutorial on ultrasound for DVT

Interpretation: Normally the veins are collapsible, failure to compress the vein (**Video**) or identification of echogenic material in the vein lumen suggests DVT (Image 18.36).

**Video:** (A) Normal compressible left Common Femoral Vein (CFV). (B) Non-compressible left CFV suggesting a DVT

### Image 18.36 DVT



*Echogenic material in the right Common Femoral Vein (CFV) indicating a DV*

Pitfalls: The 2 zone technique can only be used in ambulatory patients as studies have shown that it might miss isolated deep femoral vein DVTs which are seen in patients with prolonged immobilization.

The RUSH protocol provides a systematic stepwise approach to help rapidly identify the etiology of undifferentiated shock summarized in **video**. The sequence of

the protocol may be altered based on the clinician's assessment of the clinical condition.

**Video:** Summary of the RUSH protocol

**References and Further Reading,** [click here](#)

# BLUE protocol

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by Toh Hong Chuen

## Case Presentation

*A 68-year-old man with a history of congestive cardiac failure (CCF) and chronic obstructive pulmonary disease (COPD) presented with breathlessness and a newly productive cough for 3 days. He was non-compliant with neither medication nor fluid restriction. At triage, he dyspneic and immediately brought to the resuscitation bay. His vitals were BP 188/92mmHg, PR 119/min, RR 23/min, Temp 37.9C, SpO2 91% on 3L intranasal oxygen. Clinically, the JVP was elevated to the earlobes. Heart sounds were S1S2, breath sounds were diminished with prominent wheezing. There was mild pitting edema in the lower limbs to the knee. The diagnostic dilemma of acute exacerbation of CCF versus COPD needed to be addressed urgently.*

*While cardiac monitors and peripheral IVs were being set up, lung ultrasound was performed using the BLUE protocol.*

*Bilateral lung sliding were seen in Stage 1, negative DVT scan in Stage 2 and negative posterior lateral alveolar pleural syndrome (PLAPS) in Stage 3. This clinched the diagnosis of acute exacerbation of COPD, and he was immediately put on nebulization with salbutamol and ipratropium. IV steroids and slow maintenance fluids were started, and since he fulfilled the Anthonisen criteria for infective exacerbation, broad-spectrum antibiotics were given. The CXR performed demonstrated hyperinflated lungs, cardiomegaly, and no consolidation; while the blood tests were unremarkable. The patient improved significantly after the 3rd cycle of nebulization and did not require non-invasive ventilation. He was admitted for continued management and subsequently discharged well after 2 days.*

## Emergency

## Indication

Patients presenting with dyspnea or respiratory distress

## Contraindication

Absolute contraindication: NONE

Relative contraindication:

- Lung ultrasound should not delay immediate interventions required for recognized life threats.
- Nevertheless, lung ultrasound can often provide information that leads to the diagnosis of these life threats.

## Equipment and Patient Preparation

### Patient Preparation

- Position: Supine, or semi-recumbent
- Consent: Verbal consent is adequate
- Others: Apply universal precaution

### Equipment Preparation

Probe: Curvilinear (preferred), or linear

Preset: Lung preset if available.

- If lung preset is not available, use the abdominal preset, but switch off all B mode optimization settings (e.g., tissue harmonics)

- Others: Gel

## Procedure Steps

1. Explain the procedure to the patient and get consent.
2. Set to lung preset, and apply gel to the probe
3. Scan sequentially, and up to the 3rd stage if required.

### A. Stage 1: Anterior Chest Wall

- Four sites: Right and Left – Upper and Lower Blue Points
- Proceed to Stage 2 only if A profile (i.e., “normal”) is identified

### B. Stage 2: DVT scan

- Refer to the section on DVT scanning
- Proceed to Stage 3 only if the scan is negative for DVT (i.e., normal)

### C. Stage 3: Posterior Lateral Chest Wall

- Two sites: right/left PLAPS point.

## Blue Protocol Scanning Sites

There are many proposed lung ultrasound scanning sites. In the original paper on BLUE Protocol, the chest wall is divided into 6 zones (Anterior, Lateral and Posterior zones of the right and left chest walls), which is further subdivided into upper and lower halves (i.e., 12 sites).

Currently, the scanning sites have been simplified to the 2 BLUE Points on the anterior chest wall (stage 1) and 1 PLAPS point in the posterior-lateral chest wall (stage 3), performed on each side of the thorax.

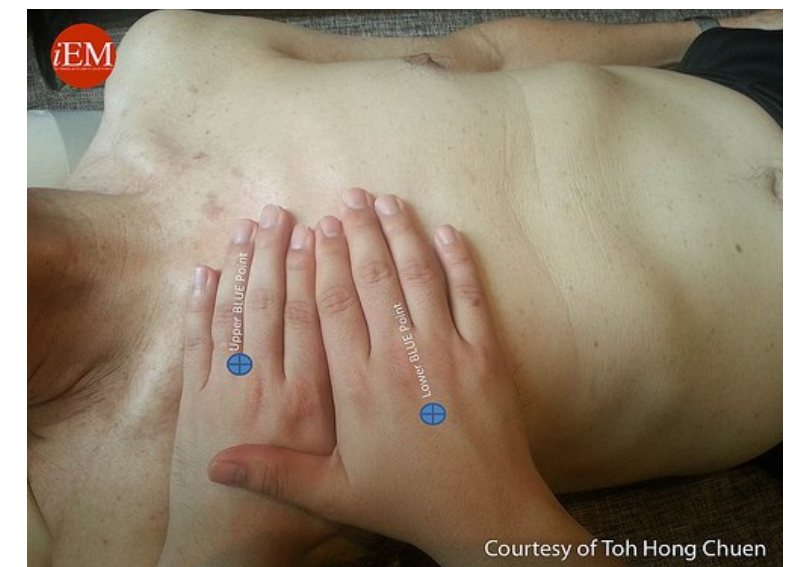
Locate the Upper and Lower BLUE (Image 18.37) and PLAPS Points (Image 18.38) as follow:

1. Put two hands side by side with index fingers touching each other (i.e., excluding the palm). The examiner hand size should approximate that of the patient’s hands.

2. Place the upper hand just below the clavicle, with the fingertips in the mid-sternum.

- Upper BLUE Point is in the middle of the upper hand (i.e., between the root of 3rd and 4th fingers)

**Image 18.37** Upper and Lower BLUE Points

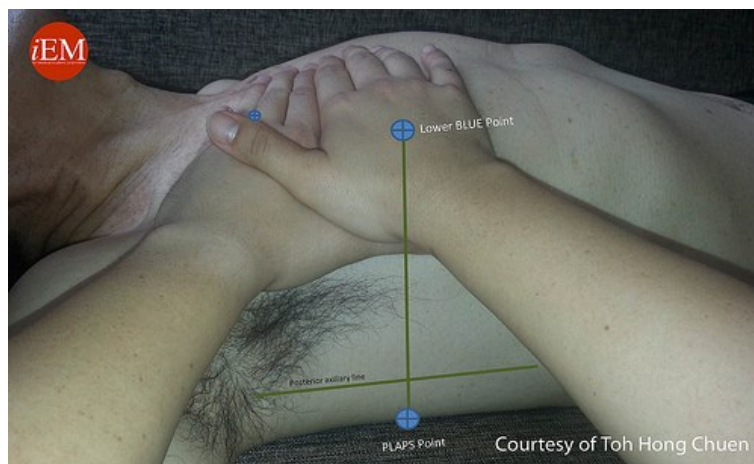


Courtesy of Toh Hong Chuen



- Lower BLUE Point is in the middle of the palm of the lower hand.
- PLAPS Point is the horizontal continuation of the lower BLUE Point, as posterior as possible to the posterior axillary line with the patient remaining supine.

**Image 18.38** PLAPS Points



## Lung Ultrasound Findings

These are the building blocks of the BLUE Protocol. As they are mostly artifacts, settings on B mode imaging which minimizes artifacts (so as to improve image resolution) should be switched off.

### Bat Sign

The bat sign is critical for correct identification of the pleural line. Always begin lung ultrasound by identifying the bat sign before proceeding to look for artifacts and pathologies.

This sign is formed when scanning across 2 ribs with the intervening intercostal space.

The wings are formed by the 2 ribs, casting an acoustic shadow. The body is the first continuous horizontal hyperechoic line that starts below one rib and extends all the way to the other. (**Video: Bat Sign**) The body is the pleural line, i.e., parietal pleural. Normally, the pleural line is opposed to and hence indistinguishable from the lung line (formed by the visceral pleura).

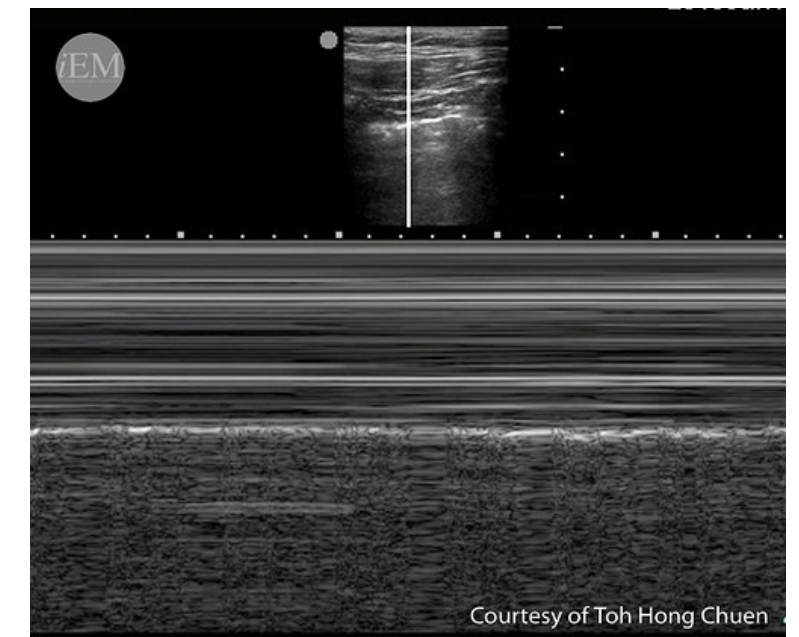
### Lung Sliding

When the lung expands and contracts with respiration, the parietal (pleural line) and visceral pleural (lung line) move and slide over each other, creating a shimmering or sparkling motion artifact

on B mode at the pleural line, termed lung sliding (**Video: Lung Sliding**).

This motion artifact produces the sea-shore sign on M-Mode (Image 18.39: Seashore Sign).

**Image 18.39** Seashore Sign



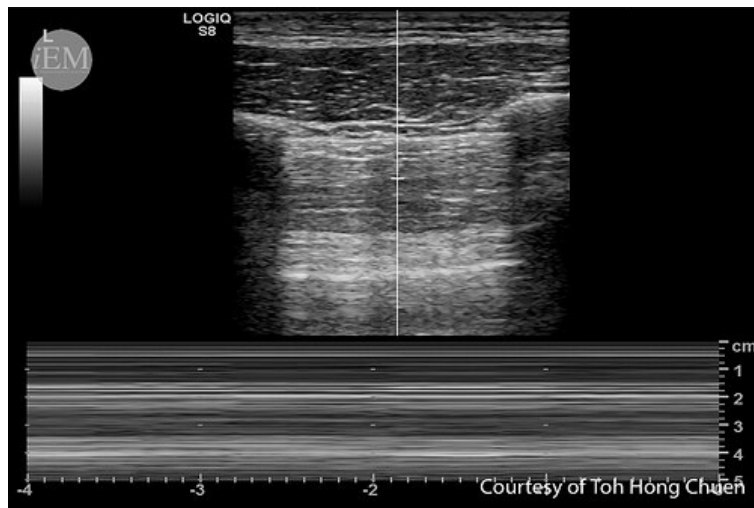
Absent lung sliding is always abnormal and occurs when the two pleural are

- Separated, for example by air in the case of pneumothorax
- Opposed but stuck to each other (pleurodesis)

- Opposed but not moving (mainstem intubation)

The absence of lung sliding is readily apparent in B mode (**Video**: Absent Lung Sliding) and produces the stratosphere sign on M-Mode. (Image 18.40: Stratosphere Sign)

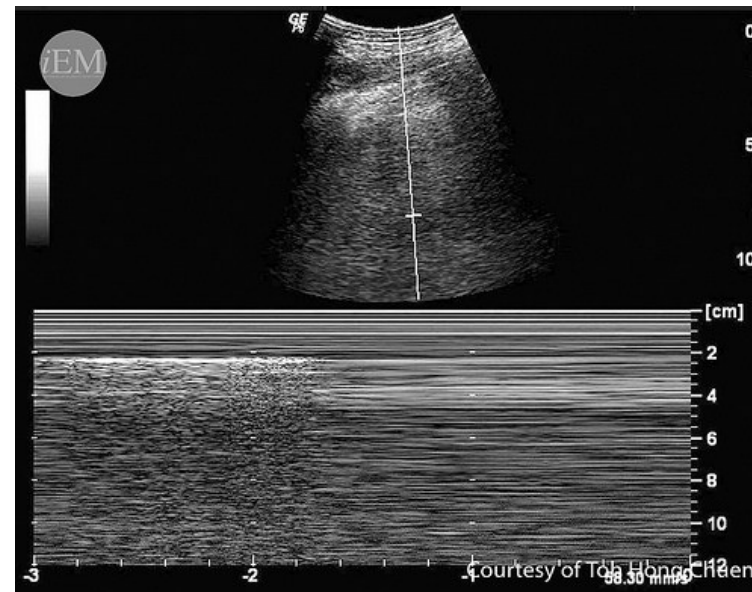
**Image 18.40** Stratosphere Sign



## Lung Point

This refers to the appearance and disappearance of lung sliding (**Video**: Lung Point on B Mode) with respiration at a specific point on the pleural. It is equivalent to having alternating sea-shore and stratosphere signs on M Mode (Image 18.41: Lung Point on M Mode).

**Image 18.41** Lung Point on M Mode



Lung Point is pathognomonic of pneumothorax.

It reflects the size of the pneumothorax (moderate if seen anteriorly, large if seen posteriorly, and total collapse if absent) and may guide the need for intervention.

Most pneumothoraces with lung point in the lateral chest wall requires chest tube (90%), compared to those with anterior location (8%).

## A-lines

These are horizontal reverberation artifacts arising from the pleural line. Consequently, they appear and are

repeated at regular intervals below the pleural line, at a distance which is equal to the distance between the probe-skin interface and pleural.

The presence of A-lines indicates good scanning technique, as the probe is perpendicular to the pleural line – a requisite for the generation of this artifact. The converse is also true. This effect is demonstrated in the clip (**Video**: A Lines), where the A lines disappear when the probe is tilted away from its initial perpendicular position.

## B-lines

B-lines are artifacts with 7 characteristics, of which the first three are always present.

They are comet-tail artifacts arising strictly from the pleural line and always move in concert with lung sliding (if lung sliding is present). They are most often hyperechoic, well defined, long and laser-like, and erases the A lines along its path. (**Video**: B Lines)

They occur when the subpleural visceral interlobular septa are edematous. This can be found in several conditions, such as acute cardiogenic pulmonary edema, ARDS, pulmonary contusion, and pneumonia.

The following terms are commonly encountered in literature but are not included in the BLUE Protocol.

Lung rockets: 3 or more B lines within a rib space. Septal rockets contain between 3-5 B lines per rib space. Ground glass rockets have 6 or more, which often coalesce to form a bright curtain-like artifact “hanging” from the pleural line.

Interstitial syndrome: bilateral anterior lung rockets. Posterior lung rockets are not considered as they may be due to gravitational pull.

## Pleural effusion

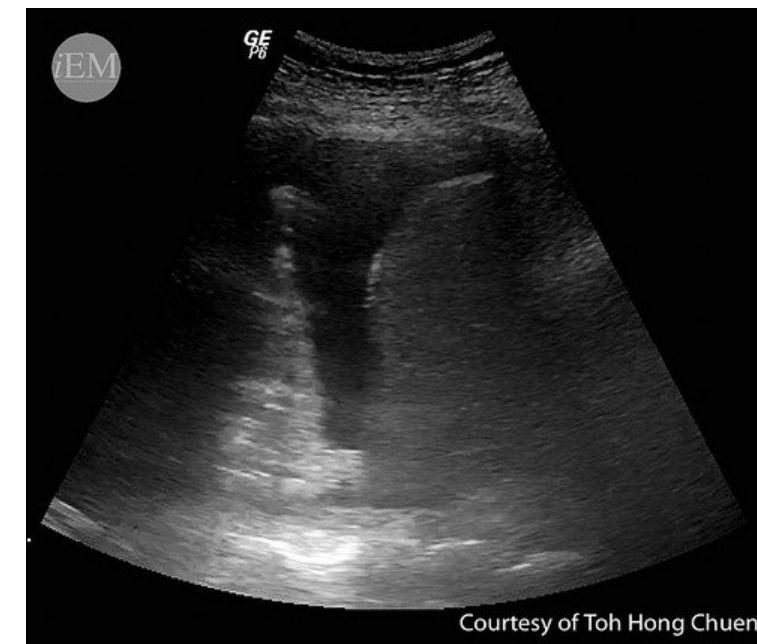
Presence of anechoic collection between the pleural and lung line (Image 18.42: Pleural Effusion).

## Consolidation

Tissue-like sign: indicating translobular consolidation (**Video**: Tissue-like Sign)

When the entire lobe is consolidated, it has a tissue-like appearance similar to the liver.

**Image 18.42** Pleural Effusion



Shred sign: indicating non-translobular consolidation (Image 18.43: Shred Sign, **Video**: Shred Sign)

The interface between consolidated and aerated lung is irregular, and appeared as if it has been “shredded.”

## Lung Profiles

The lung findings described above are used to characterize the lung profile.

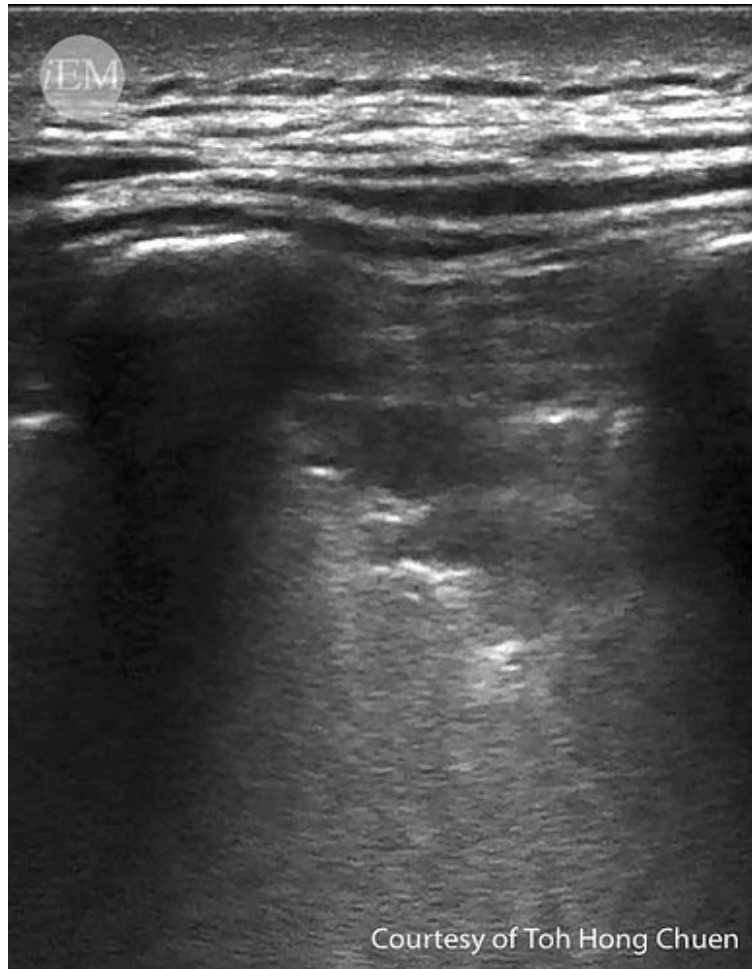
## Anterior Chest Wall

There are 6 profiles on the anterior chest wall (i.e., Upper and Lower BLUE Points)

1. A profile = Bilateral A-lines with lung sliding.



## Image 18.43 Shred Sign



2. A' profile = A lines without lung sliding
3. B profile = Bilateral B lines with lung sliding
4. B' profile = B lines without lung sliding
5. A/B profile: Half A profile on one lung and half B profile at the other

6. C profile = Shred sign or tissue-like sign (regardless of size or number)

## Lateral Chest Wall

There are 2 profiles on the posterior lateral chest wall (i.e., PLAPS Point)

1. PLAPS profile (or PLAPS positive): the presence of either pleural effusion or consolidation
2. Nude profile (or PLAPS negative): absence of pleural effusion and consolidation

## Lung Profiles of Common Respiratory Diseases

1. Cardiogenic pulmonary edema: B profile
2. COPD or asthma: A profile with negative DVT scan, negative PLAPS
3. Pulmonary embolism: A profile with positive DVT scan
4. Pneumothorax: A' profile with a lung point

5. Pneumonia: Variable: A + PLAPS profile, A/B profile, B' profile & C profile

There are two important caveats in relating lung profiles to specific diseases:

1. The normal non-pathological lung has the same findings as patients with COPD or asthma, i.e., A profile with negative DVT scan and negative PLAPS.
2. Patients with A' profile and no lung sliding requires additional imaging modalities
  - This could still be due to a pneumothorax (i.e., massive one with a complete collapse of the lung) or other rare causes (e.g., pleurodesis).

## Hint and Pitfalls

- The BLUE protocol is not an acronym. It highlights the indication (and utility) for using the protocol, i.e., a patient who is "blue" from respiratory distress. It is a rapid and efficient way of diagnosing



the 5 major respiratory diseases, with a reported accuracy of 90.5%.

- Perform lung ultrasound immediately after clinical examination, prior to CXR. It yields diagnostic information rapidly and can expedite treatment.
  - Recognizing the B profile, for example, takes only less than 10 seconds.
  - Completing the entire protocol (i.e., up to Stage 3) requires less than 3 minutes.
- Stay focus and scan only the BLUE points and PLAPS points.
  - Other sites can be scan when the time is available.
- Always interpret ultrasound findings in the context of clinical findings; and integrate both in the clinical decision-making process.
- Pitfalls of the BLUE Protocol:

- It cannot be used for patients with mixed or multiple respiratory disorders.
- It does not identify rare respiratory disorders (defined as occurring with a frequency of <2% of ICU patients in the single center that was studied)
- Massive pleural effusion is not included in the protocol, though diagnosis is not an issue)
- It cannot be used for non-respiratory causes of breathlessness, e.g., hyperventilation from metabolic acidosis or profound anemia.
- It is not designed to provide information on the patient's hemodynamic status.
  - This could also be performed using point of care ultrasound, by integrating the focused cardiac and IVC with

the lung findings, in the form of the FALLS protocol.

## Post Procedure Care and Recommendations

None

## Complications

None

## Pediatric, Geriatric, and Pregnant Patient Considerations

### Geriatric

The BLUE protocol is derived from a study of 301 consecutive adult patients and is applicable to the geriatric population.

### Pregnant

While there are no pregnant patients which are reported in the original paper<sup>2</sup>, the principles in the diagnostic algorithm are applicable in pregnancy.

### Pediatric

In the same way, the BLUE protocol can also be adapted for use in the pediatric patients.

**References and Further Reading,** click [here](#)

# How to Read C-Spine X-Ray

---

by Dejvid Ahmetović and Gregor Prosen

## Introduction

C-spine x-ray interpretation is one of the fundamental skills of emergency physicians. Although current guidelines lead us to use CT scan for a suspected c-spine injury, c-spine x-rays are still valuable in some low resource settings and patient groups who are susceptible to radiation. Therefore, this chapter will summarize the basics of c-spine x-ray interpretation.

Interpretation of radiographs has its limitations, which more or less depending on the individual's knowledge of anatomy and clinical experience.

Because anatomical landmarks for measurements can sometimes be difficult to find or identify. A more systematic approach to reading cervical radiographs can significantly reduce the chances of missing an important injury.

## Visualisation

Plain radiographs, when they show the lateral projection of the cervical spine and include an open mouth view, are fairly sensitive in identifying c-spine fractures. The risk of missing a significant fracture is, according to statistics, less than 1%. Addition of the anteroposterior (AP) projection increases sensitivity to approximately 100%. All of the three essential above mentioned projections can be seen in Image 18.44.

## Image 18.44 C-spine essential views



Lateral view with normal slight lordosis (A), Odontoid or open mouth view of the atlas and axis (B), Standard anteroposterior or AP view with open mouth, it can also be taken with closed mouth (C).

Before analyzing cervical radiographs, some additional facts need to be presented.

Most spinal injuries occur at the junctions of the spine: craniocervical, cervicothoracic, thoracolumbar and lumbosacral.

Only c-spine radiograph one should be satisfied with is the one showing all of the 7 cervical vertebrae (C1-Th1).

The C7-Th1 vertebrae may be obscured in muscular or obese patients (Image 18.45), or in patients with spinal cord lesions that affect the muscles which normally depress shoulders. Such lesions that leave the trapezius muscle unopposed occur in the lower cervical region. Shoulders can be depressed by pulling the arms down slowly and steadily, or if the patient is capable, asking them to depress one shoulder and lift the other hand above his

## Image 18.45 Inadequate c-spine lateral x-rays



Two examples of a cervical x-ray that is not good enough for the evaluation of the possible injury of the neck.

head to achieve the swimmer's position, which better visualizes the lower vertebrae.

### There are 3 basic views of c-spine

1. Cross-Table Lateral View
2. Odontoid – Open Mouth View
3. Anteroposterior View

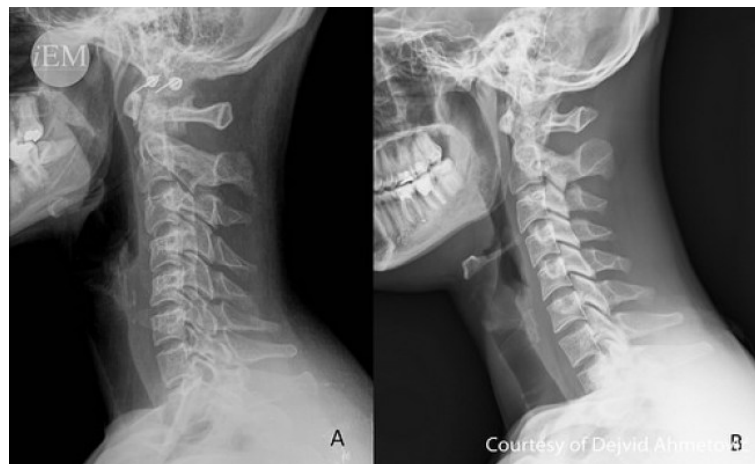


## Cross-Table Lateral View

The lateral (cross-table) view is the most helpful x-ray study in diagnosing c-spine injuries. Inspection of the x-ray should be thorough, methodical and complete. At this point it is not easy to differentiate ‘ABCs’, because of all the acronyms across the field of medicine, but the ‘ABCs’ in this case stands for: A – alignment and adequacy, B – bone abnormalities, C – cartilage space assessment and S for soft tissues.

### A – Alignment and adequacy

#### Image 18.46 Alignment and adequacy



Example of a slightly rotated not ideal lateral projection of the cervical spine in (A) and an x-ray of an ideal lateral projection in (B).

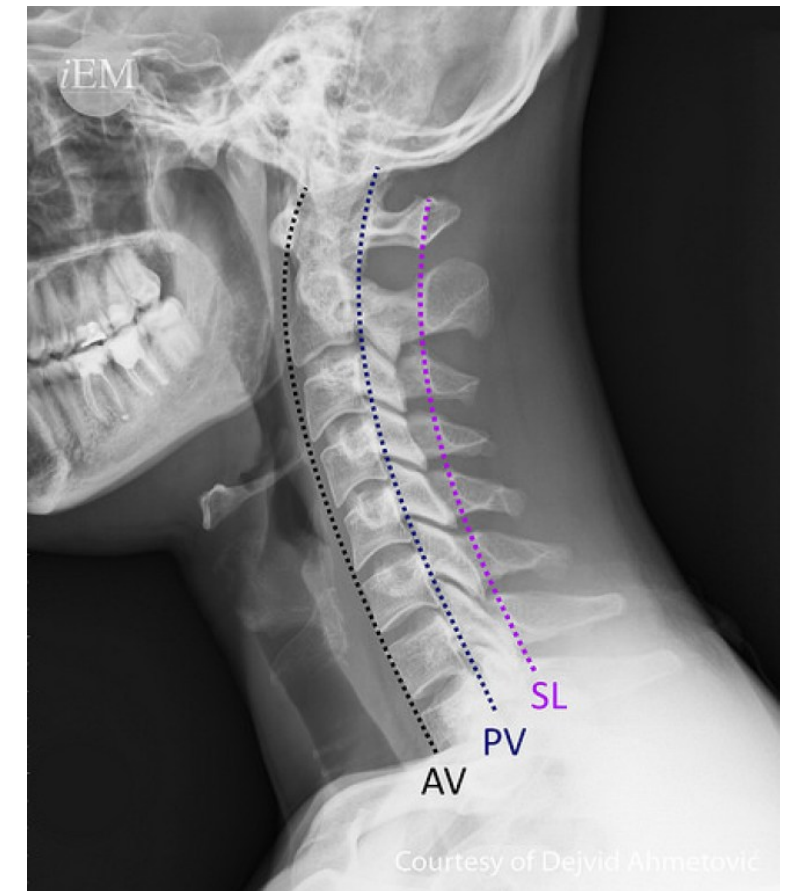
First, visualize the spine from the base of the skull to the C7-Th1 junction. Next, check if the x-ray is a real lateral view, or if it is slightly rotated. Facet joints are best visualized when we have a proper lateral projection. (see Image 18.46).

To check for proper alignment, look for a normal smooth lordotic curve and imagine two lines, each running along the anterior and posterior margins of vertebral bodies. Additionally, a third line (spino-laminar line), running along the base of spinous processes and up to the posterior aspect of the foramen magnum, must be visualized (Image 18.47).

All three lines should form a smooth and lordotic curve of the cervical spine. Any disruption in the flow of these lines suggests either a bony or a ligamentous injury (Image 18.48).

An exception to this rule is a pseudo-subluxation of C2 and C3 in the pediatric population, which can cause confusion. In these cases inspect the spino-laminar line from C1-C3 and be suspicious of

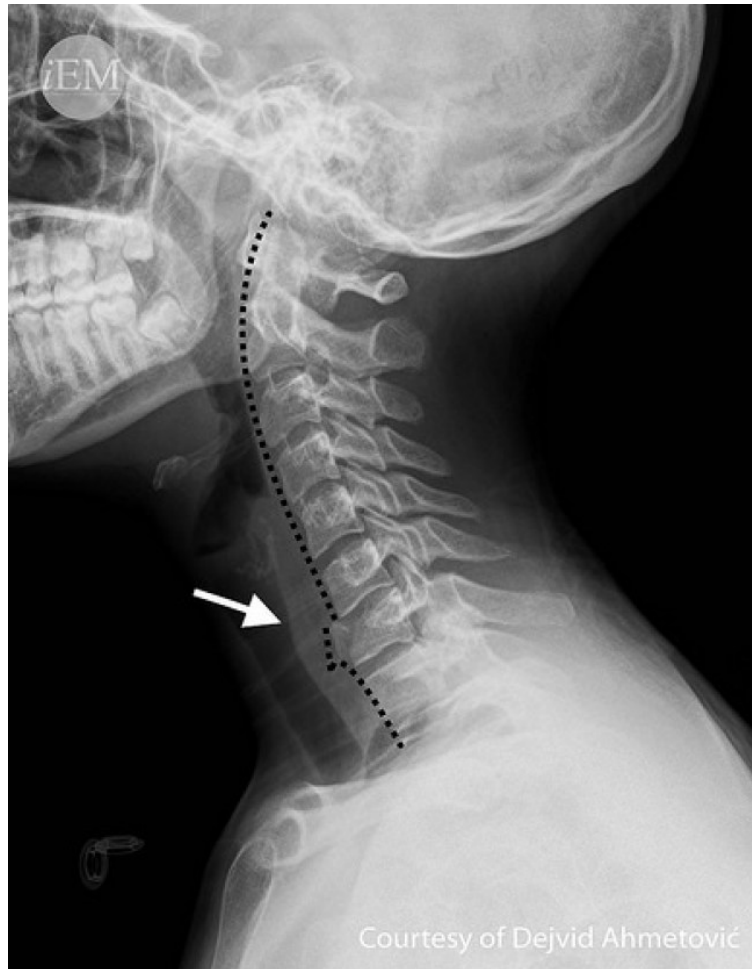
#### Image 18.47 Alignment and lines



Always assess (AV) anterior vertebral, (PV) posterior vertebral and (SL) spinolaminar lines, they should run smooth, without any disruptions, and should form a slight lordotic shape.

injury if the C2 spinous process base lies more than 2 mm from this line. Also correlate with the soft tissue findings (see below, under “S”). Furthermore, on the lateral view, inspect the predental space, which is the distance between the anterior surface of the odontoid process and the posterior aspect of the anterior

**Image 18.48** Lorem Ipsum dolor amet, consectetur



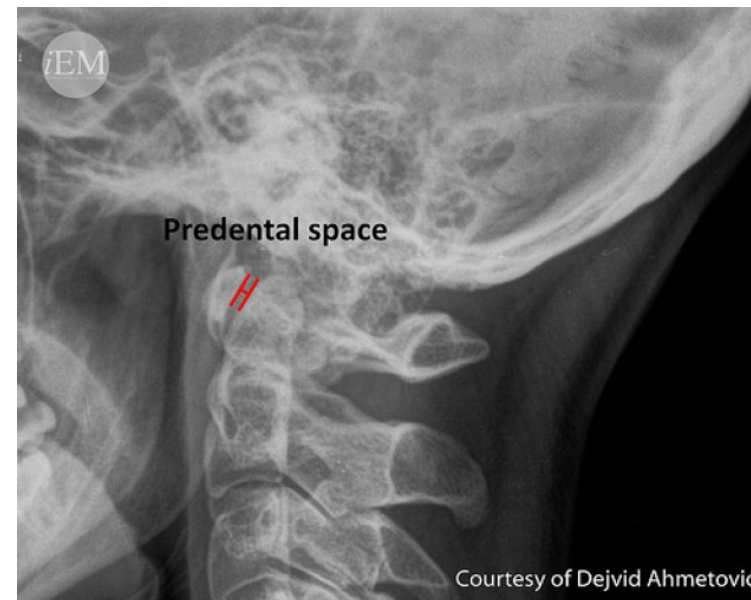
*Disruption in the shape of the AV line, that indicates injury, and in this case a fracture of the body of C7.*

ring of C1. It should not exceed 3 mm in adults or 5 mm in children. (Image 18.49).

## B – Bone

Watch for a normal bony outline of the vertebrae and bone density. Subtle changes in bone density should be noted,

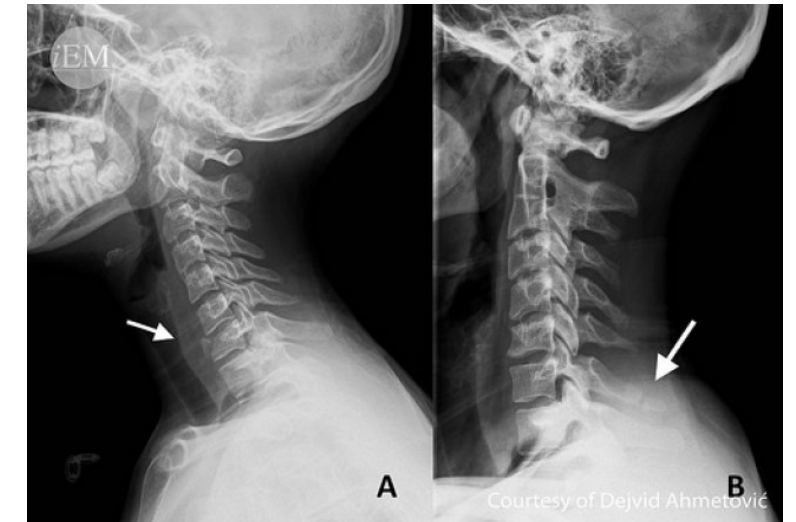
**Image 18.49** Predental space



*Predental space, the distance between the anterior surface of the odontoid process and posterior aspect of the anterior ring of C1, in adult, it should not exceed 3 mm, or 5 mm in children.*

as it may indicate a compression fracture. Areas with decreased bone density which may be found in patients with rheumatoid arthritis, osteoporosis or metastatic osteolytic lesions, are more prone to breaking under stress. Acute compression fractures of the above-mentioned changes show as areas of increased bone density (Image 18.50).

**Image 18.50**



*Watch for a non-disrupted bony outline. Disruption, as in the above examples means fracture of the bone structure. Also search for any hypo- or hyper-dense areas in the bone, as it may be the only indication of the compression fracture. In (A) slight widening of the soft tissue is visible just in front of the fracture, under the white arrow, which may indicate that this is an acute injury.*

## C – Cartilage space assessment

Inspection of a good quality lateral view x-ray in a healthy person should show uniform intervertebral spaces. (Image 18.51).

An emergency physician may diagnose subluxations and dislocations of the facet joints through the assessment of cartilage space between corpora of vertebrae, facet joints, and space between spinous



**Image 18.51** Cartilage space



*Uniform intervertebral cartilage spaces, also facet joints must be inspected, for any unusual alignment or increased space.*

processes. Increased interspinous distance by more than 50% suggests a ligamentous injury and the protective muscle spasm may make the interpretation difficult.

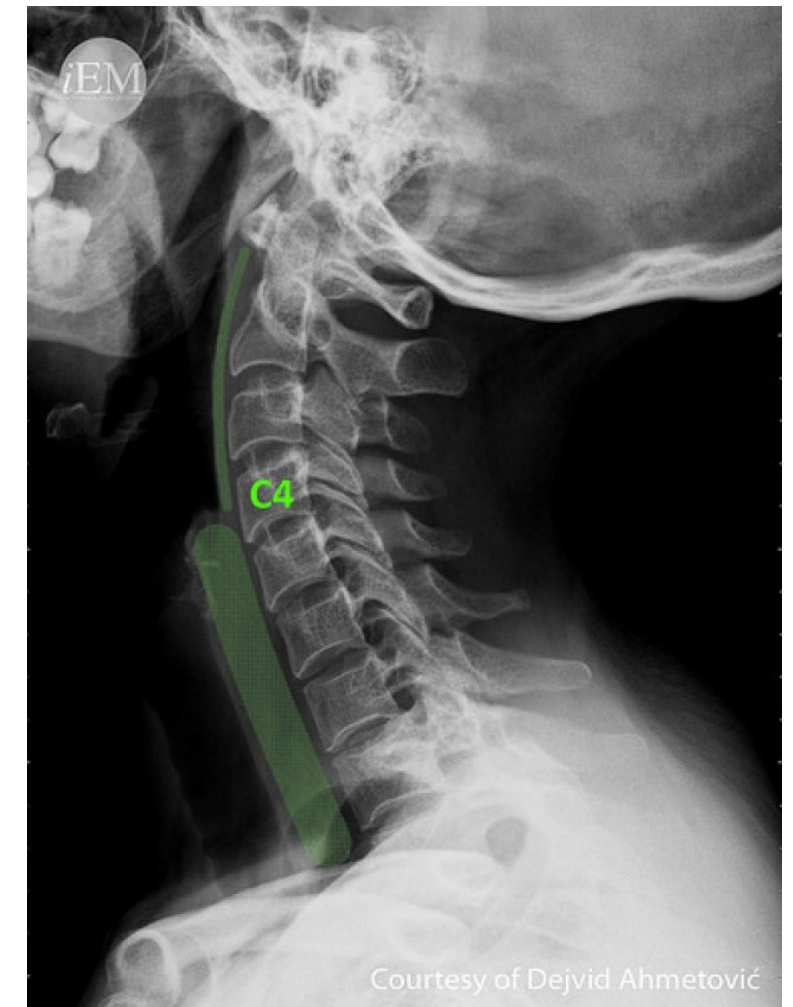
## S – Soft tissues

The prevertebral soft tissues can be used as an indicator of an acute swelling or hemorrhage resulting from an injury, and may sometimes be the only indicator of an acute injury on an x-ray. The normal width of the prevertebral tissue decreases down from C1 to C4 and increases from C4 downwards. Normal measurements from C1 to C4 are less than 7 mm (less than half of the vertebral body at this level), and less than 22 mm below the C5 (less than the vertebral body at this level) see Image 18.52. Air within soft tissue could suggest rupture of the esophagus or trachea.

## Odontoid – Open Mouth View

This is usually the second standard view obtained in the emergency department. The main goal is to picture the odontoid process of the C2 and the C1. It can be done with the mouth either open or closed. Two things are assessed when inspecting the odontoid x-ray: the distance between the odontoid process

**Image 18.52** Soft tissues

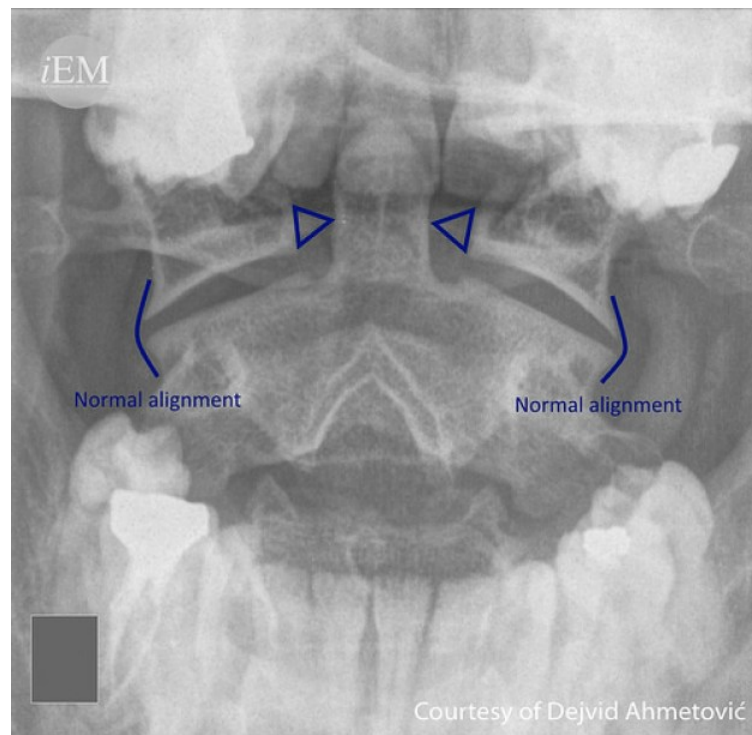


*Retro-pharyngeal soft tissue, narrows down from C1 to C4, and should not exceed more than 7mm (less than third of the vertebral body). Below the C4 soft tissue starts widening, but should not exceed 22mm (for easier thinking, should not exceed the width of the body of the vertebrae).*

and the lateral masses of the C1 should be equal. If not, the inequality may be due to a slight rotation of the head. Secondly, and considering the previous point, the

margins of C1 and C2 should remain aligned (Image 18.53).

**Image 18.53** Odontoid - Open Mouth View



*The distance between the odontoid process and the lateral masses of the C1 should be equal, if not inequality may be due to the slight rotation of the head. (If the patient has the upper central incisor teeth, we can check if the space between those two teeth aligns with the middle of the odontoid process, this might give the slight idea about rotation in case process itself is not broken and misaligned). Even with the slight rotation of the head we can still check alignment by looking at the lateral margins of the C1 and C2, which should remain aligned.*

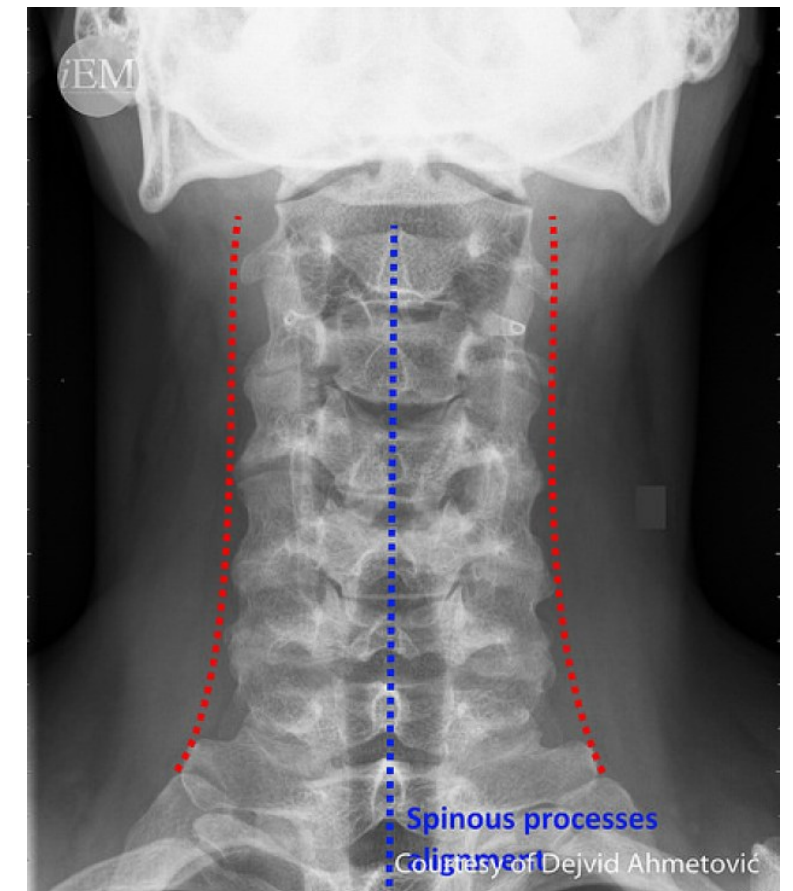
## Anteroposterior View

Images taken in this projection are usually much less clear than the two mentioned above. The tips of the spinous processes should lie in a straight line in the mid-line and distances between the spinous processes should also be checked. Anomalies, such as bifid spinous processes, can make interpretation difficult. The laryngeal and tracheal shadows should align down the middle. The alignment of the lateral masses of the vertebra should also be checked (Image 18.54).

## Other Views

Oblique and flexion/extension views are useful only to an experienced physician. Flexion and extension are often either contraindicated because of the suspected unstable trauma or impossible to accomplish because of the spastic musculature post-injury. (Image 18.55). Unsupervised or even forced flexion or extension in a patient with ligamentous injury may also lead to neurologic injury.

**Image 18.54** Anteroposterior View



*Blue line connects the spinous processes, they should lie mid-line and have an equal amount of space between. Red-line should smoothly connect the lateral masses of the vertebrae. Always check the edges of the picture, in most cases, apexes of the lungs are visible, check for pneumothorax.*

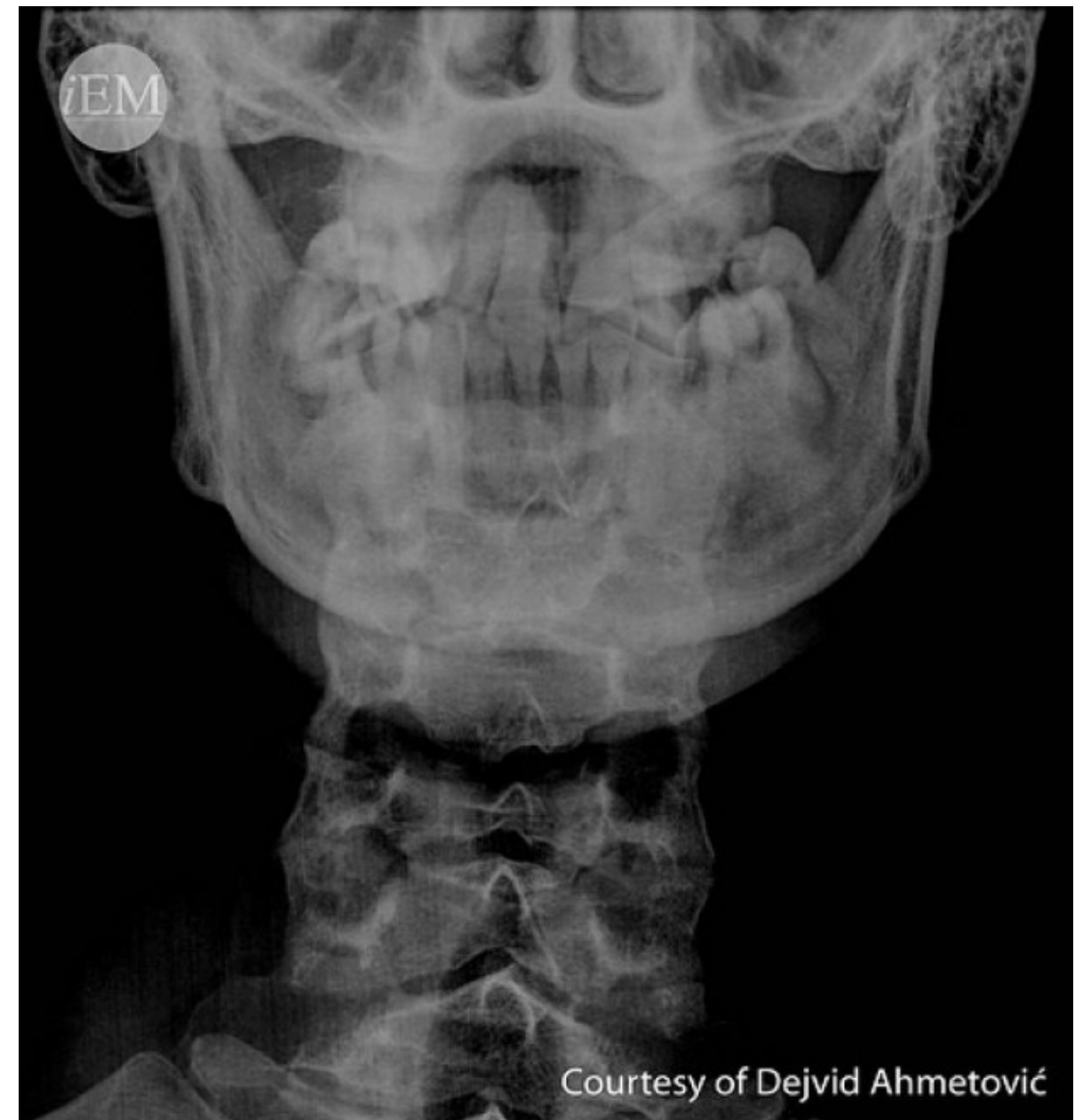


Image 18.55



*Straightened normal lordotic curvature of the c-spine, may be due to the muscle spasm as a protective mechanism, what also makes flexion and extension views hard to capture.*

Image 18.56



*Suspected fracture of the odontoid process, but with closed mouth teeth might affect the view.*

**Image 18.57**

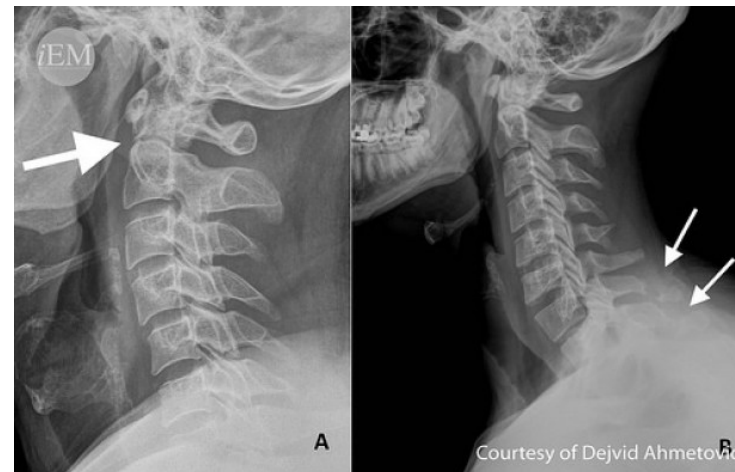


Same patient as in Figure 13, but with open mouth view, and the fracture through the body of C2 is visible, also note misalignment of lateral borders of C1 and C2 and difference in space between odontoid process and lateral masses of C2 on both sides.

## SCIWoRA (Spinal Cord Injury Without Radiographic Abnormality)

Plain radiographs are negative in 25% of pediatric patients with an injury to the spinal cord. Tenderness of the neck and careful neurologic examination must stay the main way of diagnosing a patient, especially in the pediatric population.

**Image 18.58** Lorem Ipsum dolor amet, consectetur



Lateral view of a type 2 odontoid process fracture seen in A. Fracture of spinous processes of C7 and Th1 vertebrae named Clay – shoveler fracture in B.

Even in adults, a normal cross-table lateral x-ray does not exclude a spinal cord injury. If in doubt, treat as if there is spinal cord injury until proven otherwise. It is also worthwhile to memorize a short mnemonic for children: SCIWoRA (Spinal Cord Injury Without Radiographic Abnormality).

**References and Further Reading**, click [here](#)

# How to read chest x-rays

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by Ozlem Koksai

## Introduction

Chest X-ray interpretation is one of the fundamental skills of every doctor. Emergency physicians are particularly exposed to various chest x-rays during a regular shift. Therefore, knowing the basics and pathologies in the ED setting is very important. This chapter will summarize the basics of chest x-ray interpretation and give some pathologic examples.

### There are 3 types of chest films;

- AnteroPosterior (AP)
- PosteroAnterior (PA)
- Lateral

The ideal timing can be defined as the end of inspiration, and the patient should hold his breath at that time.

Meanwhile, the X-ray tube should be 180 cm away. Unfortunately, the majority of the patients may not fit the ideal situation because of their acute problems. Emergency physicians interpret many portable (bedside) anteroposterior chest x-rays with poor quality, without lateral views to make the diagnosis. The image quality is one of the most important things in image interpretation.

## Assessing The Image Quality

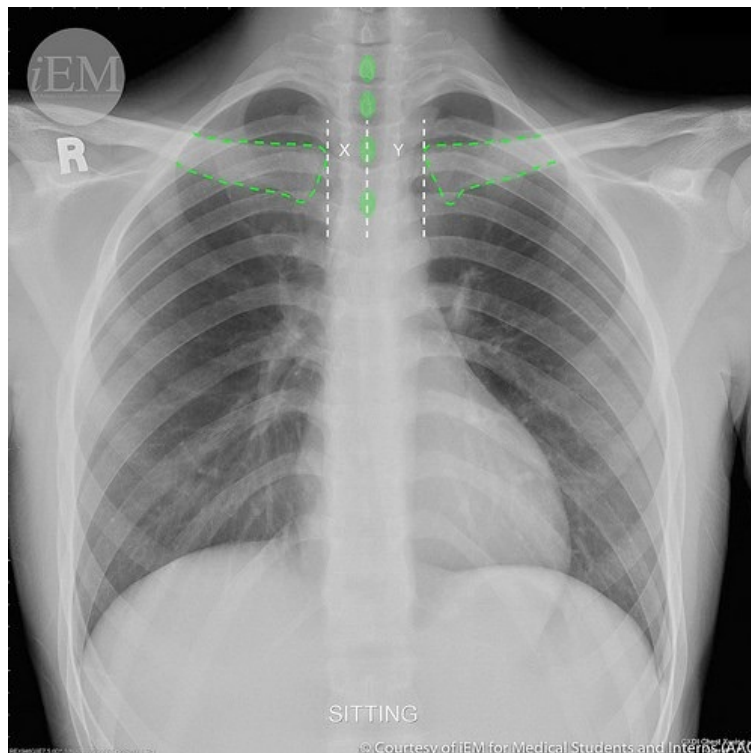
“**RIPE**” mnemonic is used; **Rotation, Inspiration, Position, Exposure(Penetration)**.

**Rotation:** The clavicles should appear symmetrical and be seen as equal length. The distance between the thoracic spinal process and clavicular heads should be equal (Image 18.59). If



there is a rotation, mediastinum may look abnormal.

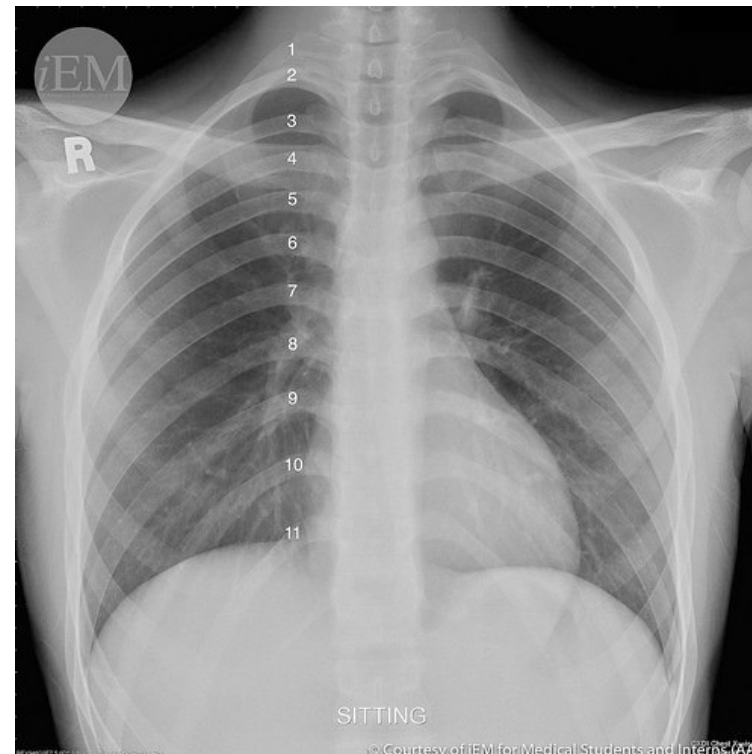
**Image 18.59**



*The clavicular heads and spinous process alignment. The x-ray shows minimal rotation. Compare X and Y.*

**Inspiration:** On good inspiration, the diaphragm should be seen at the level of the 8th – 10th posterior rib or 5th – 6th anterior rib.

**Image 18.60**

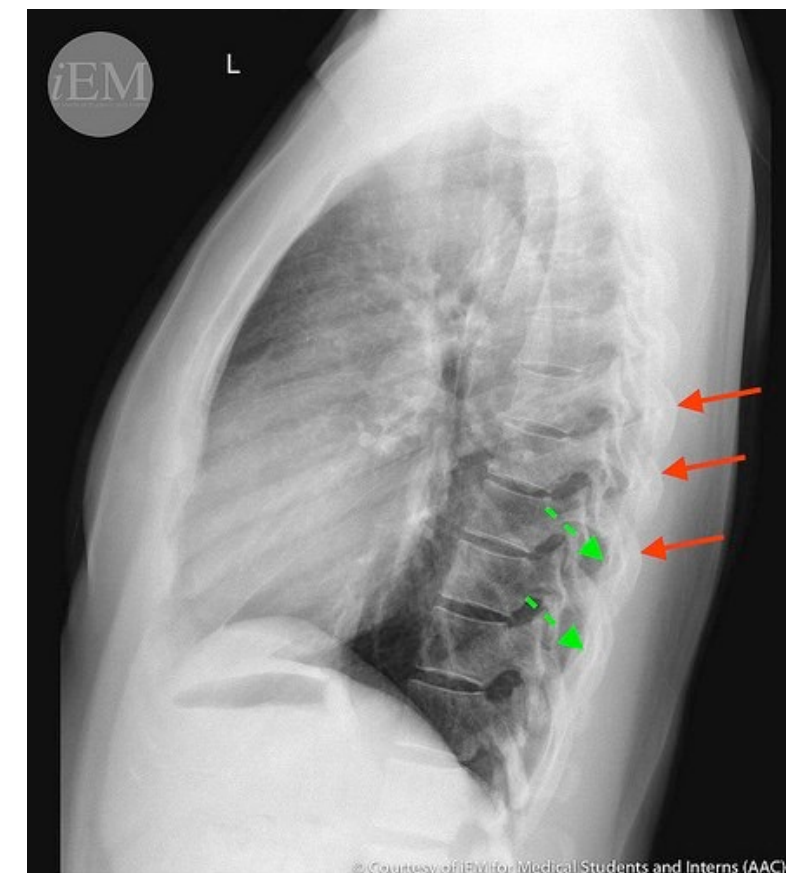


*The chest x-ray shows adequate inspiration.*

**Position:** PA, AP, or lateral view? The standard chest X-Rays consists of a PA and lateral chest X-Ray.

The normal lateral chest x-ray view is obtained with the left chest against the cassette. If the x-ray is a true lateral, the right ribs are larger due to magnification and usually projected posteriorly to the left ribs (Image 18.61).

**Image 18.61**



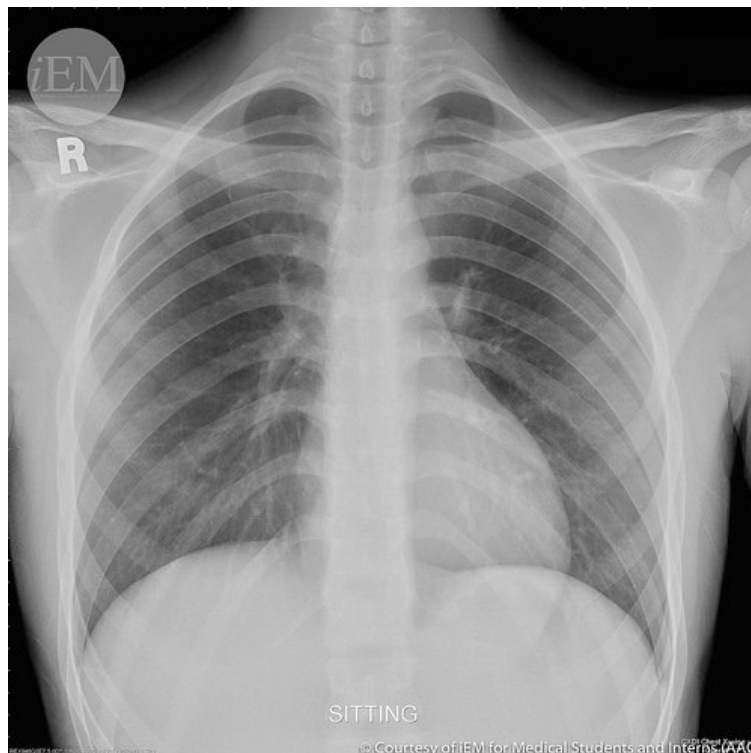
*The right ribs (red arrows) and left ribs (green arrows) on the lateral chest X-Ray.*

On the AP film, the chest has a different appearance. The heart and mediastinal shadow are magnified because of anterior structures, mainly sternum. This view is taken mostly at the bedside as portable. Some patients are at semi-erect or supine position. Therefore, mediastinal structures are widened because of gravity.

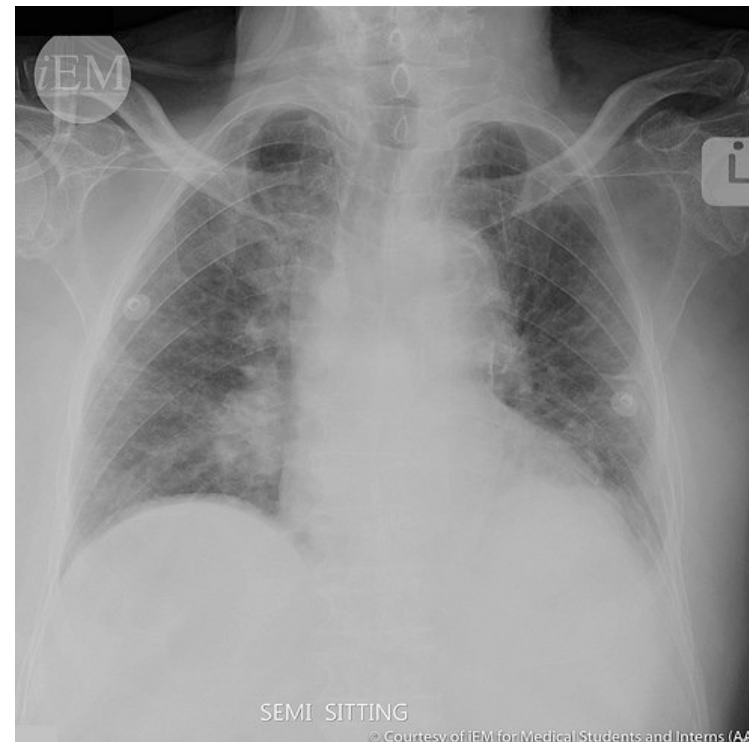


The pulmonary vasculature is altered when patients are examined in the supine position. The size of the pulmonary vasculature is more homogeneous throughout the upper and the lower lobes. (Image 18.62 and Image 18.63). Supine views are less useful and should be reserved for critical patients who cannot stand erect position.

**Image 18.62** The normal X-Ray film



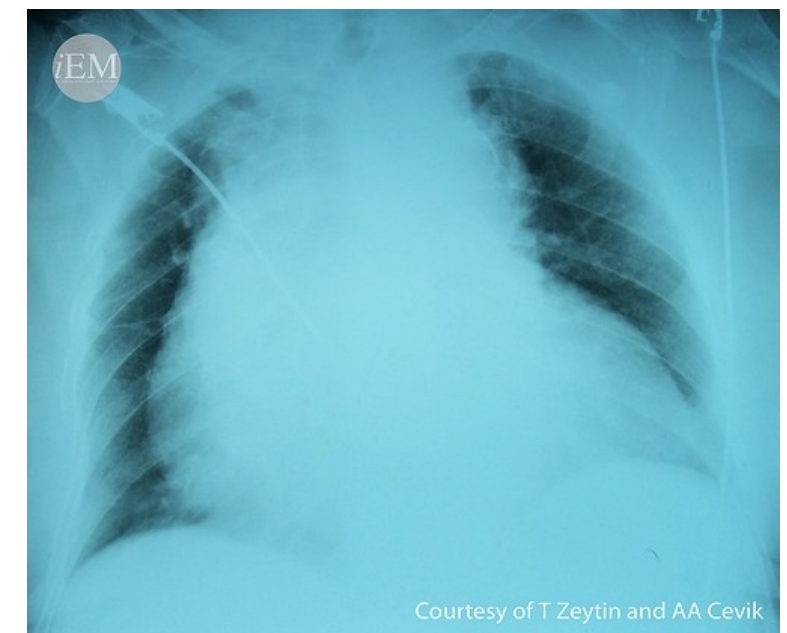
**Image 18.63**



*The AP X-Ray shows magnification of the heart and widening of the mediastinum.*

**Exposure / Penetration:** Ideally, you should be able to see the heart, the blood vessels, and the intervertebral spaces. Exposure should be adequate if you are able to see approximately T4 vertebra and spinal process. If the film is underexposed, you will not be able to see them (Image 18.64). If the film is overexposed, details of bone structures will be lost (Image 18.65).

**Image 18.64**



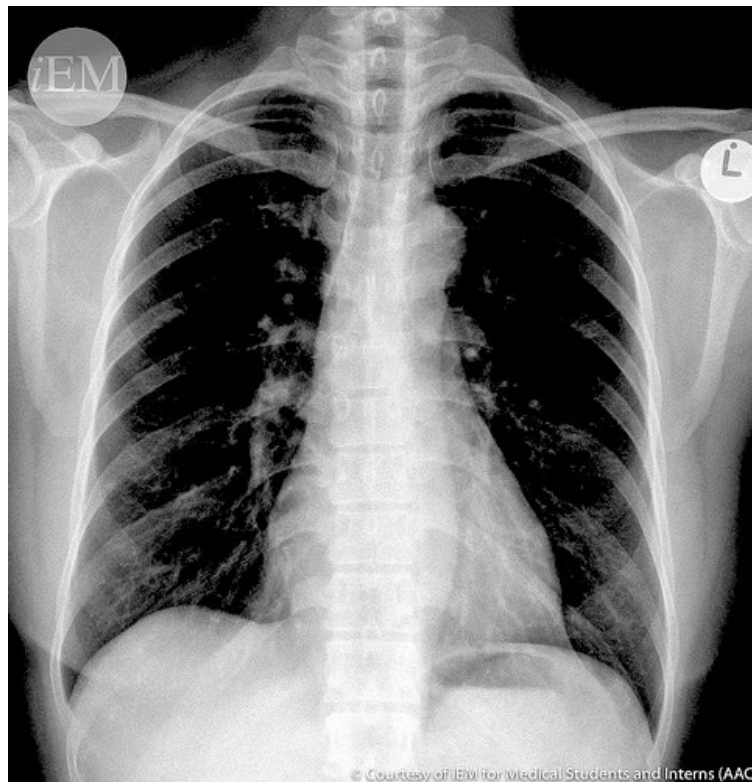
*Underexposed PA X-Ray film. You can not appreciate thoracic vertebrae.*

## Interpretation

The interpretation of a chest X-Ray should be approached systematically. For chest X-Rays, there is a classic schematic: “ABCDEF.” You should first check the patient’s name and date of the film. You should also check the side marker, and the film position (PA or AP). Finally, you should check patient’s position such as supine, erect or semi-erect.

**The analysis is ABCDEF:**

## Image 18.65



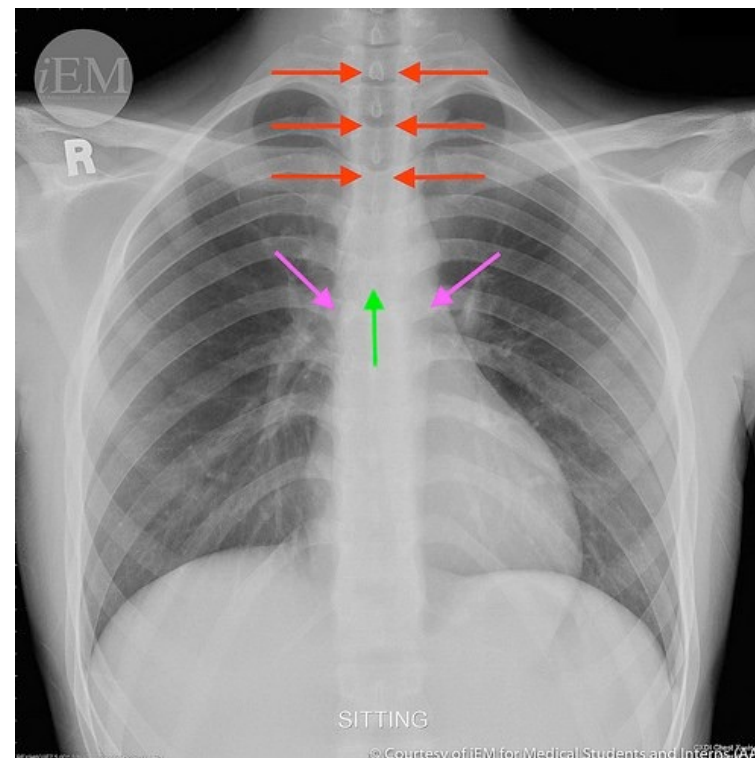
Overexposed PA X-Ray film. You are able to see all vertebral bodies with obvious intervertebral spaces.

- Airways
- Bones
- Cardiac
- Diaphragm
- Extrathoracic tissues
- Fields and Fissures

## A – AIRWAY

The trachea, carina and both main bronchi are called the upper airway and should all be visible on an AP view (Image 18.66).

## Image 18.66



Airway structures on the chest X-Ray. (Red Arrows: trachea, Green Arrow: carina, Pink Arrows: left and right main bronchus)

Look for if there is any deviation of the trachea away from the midline. Introduction of air into one side of the chest cavity will cause that side of the lung to collapse. The collapsed lung will

push the trachea to the opposite side and resulting in a deviation that will show up on chest X-Ray.

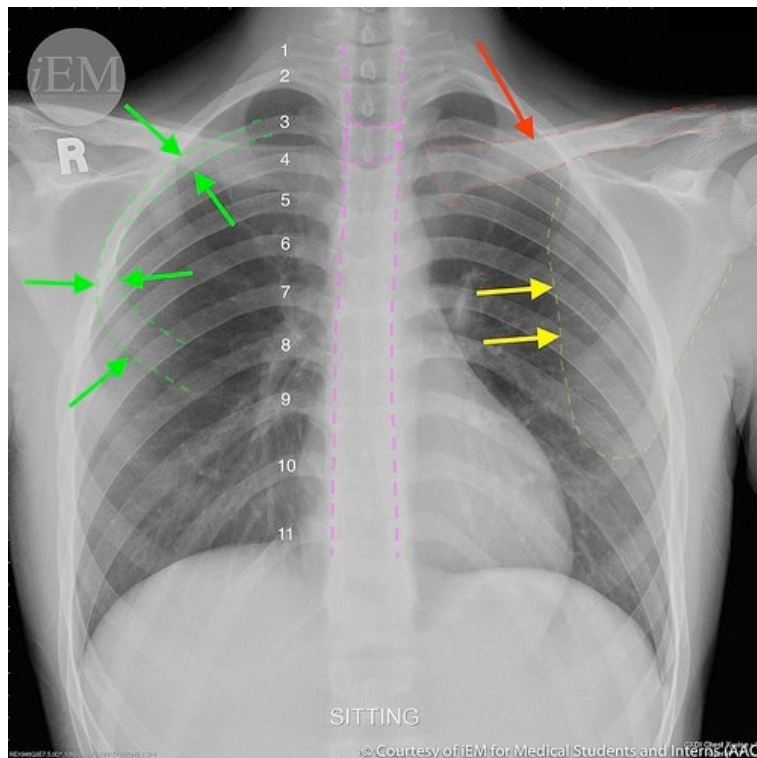
## B – BONES

A chest X-Ray provides a good view to look for ribs and clavicle fractures. Clavicular fractures are usually at the middle 3rd of the clavicle, which is easy to see in chest X-Rays. Rib fractures, however, can sometimes be hard to see. Each rib should be followed across its length to look for fracture lines or step-offs that could indicate a fracture.

Hyperinflated lungs are seen as the result of chronic obstructive pulmonary disease where the patient is unable to fully expel the air that is inhaled with every breath. Because of this, overinflation will result in a greater number of ribs that can be visible on the chest X-Rays. Normally, 8-10 ribs are expected to be seen on the chest X-Ray (Image 18.67).



**Image 18.67**



*Bone structures on the PA chest X-Ray. (Numbers: ribs, red dashed line and arrows: clavicle, yellow dashed line and arrows: medial border of scapula, green dashed line and arrows: 3rd rib, pink dashed line: vertebras)*

## C – CARDIAC

This part involves the heart and surrounding structures. The silhouette of the heart should be identified, and the heart borders should be clear. As a general rule, the heart base should not be wider than 1/2 the total width of the diaphragm. If the heart base is 1/2 the width of the diaphragm on the chest X-

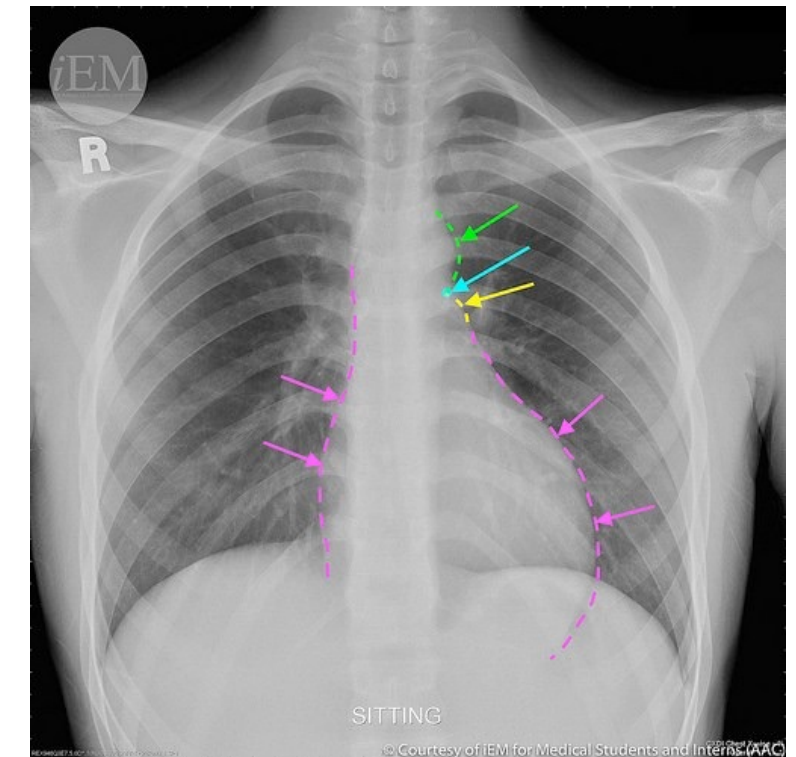
Ray, it refers to cardiomegaly or pericardial effusion.

The aortic arch and the left pulmonary artery should be visible as two semi-circles above the left atrium. There is a space called the “Aortopulmonary Window” that has the following borders: ascending aortic arch (anterior), descending aortic arch (posterior), left pulmonary artery (inferior), inferior border of aortic arch (superior). The window should be “concave” in the lateral border (Image 18.68). If it is not, mediastinal lymphadenopathy and aorta/pulmonary artery aneurysms are possible. The left hilar point is slightly higher than the right hilar point. The hilar point should be at the level of the lateral extent of the right 6th rib. The inferior vena cava lies end of the right cardiophrenic angle. The structures should be visible behind the heart especially the spine, paraspinal region and azygoesophageal line.

In ideal circumstances, mediastinum is maximum 6 cm in a PA chest x-ray, and

further investigation is considered if it is more than 8 cm.

**Image 18.68**

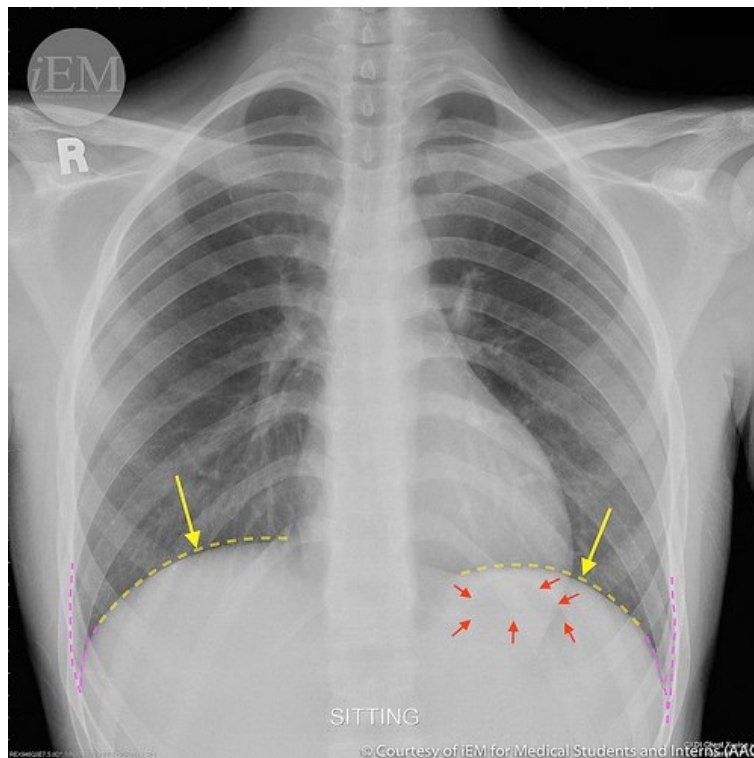


*Heart borders on the AP chest X-Ray. (Pink dashed lines and arrows: heart borders, Yellow dashed line and arrow: Aortic Arch, Blue circle, and arrow: Aortopulmonary Window)*

## D – DIAPHRAGM

The outline of the diaphragm should be clear and smooth. Right hemidiaphragm should be higher than the left (Image 18.69). It has 3 major characteristics that can be found on chest X-Ray:

**Image 18.69**



The view of the diaphragm on the AP chest X-Ray. (Yellow dashed lines and arrows: diaphragm, red arrow: gastric air bubble, pink dashed lines: costophrenic angles)

angle is closer to 90 degrees, then the lungs could be hyperexpanded (e.g., COPD) and be pushing the diaphragm down into the abdomen. If the costophrenic angle is blunting, that usually is indicative of pleural effusion.

## E – EXTRATHORACIC TISSUES

Mostly this means as the lung parenchyma. Lung fields can be divided into zones: upper, middle, and lower zones (Figure-12);

- Upper zone: from the apex to 2nd costal cartilage.

1.The gastric air bubble on the left.

2.The diaphragmatic contour looks like a “dome” shape, and the right side located little higher than the left.

3.The costophrenic angle is the lateral point of attachment for the diaphragm, and it should be a clear, sharp, and a triangle-shaped at either end. If the

- Middle zone: between 2nd and 4th costal cartilage.
- Lower zone: between 4th and 6th costal cartilage.

So you should compare the lung parenchyma left to right in the upper, middle and lower zones and see whether there is a difference.

Look for equal radiolucency

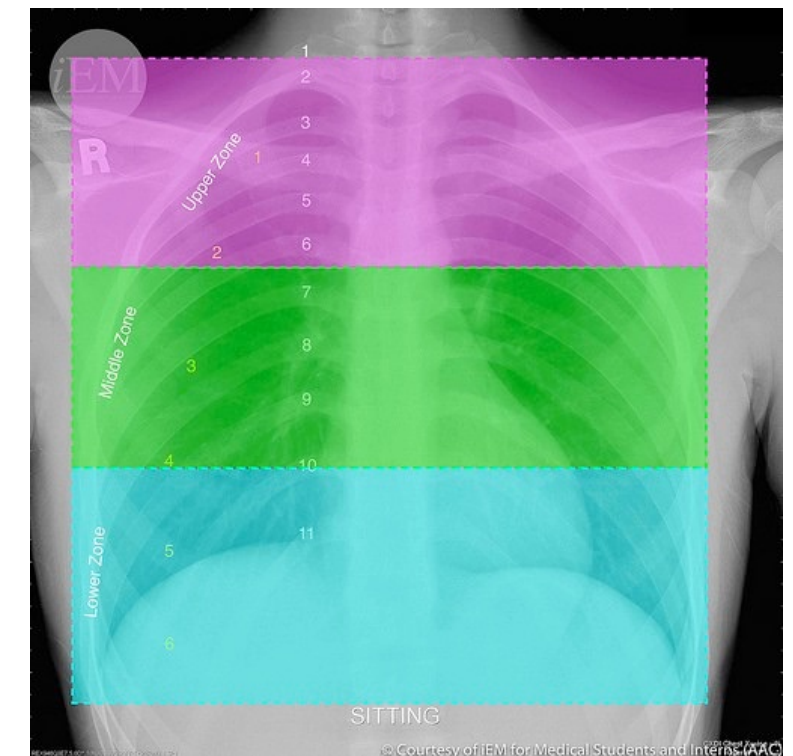
between the left and the right lungs zones. The horizontal fissure on the right divides the upper and middle lobes; from the hilum to the 6th rib at the axillary line.

You should also check soft tissues outside the thorax for subcutaneous air, foreign body, bizarre density, etc.

## F – FIELDS AND FISSURES

You should check lung fields for infiltrates. Identify the location of infiltrates and identify the pattern of infiltration (interstitial or alveolar pattern). Look for air bronchograms, nodules, Kerley B

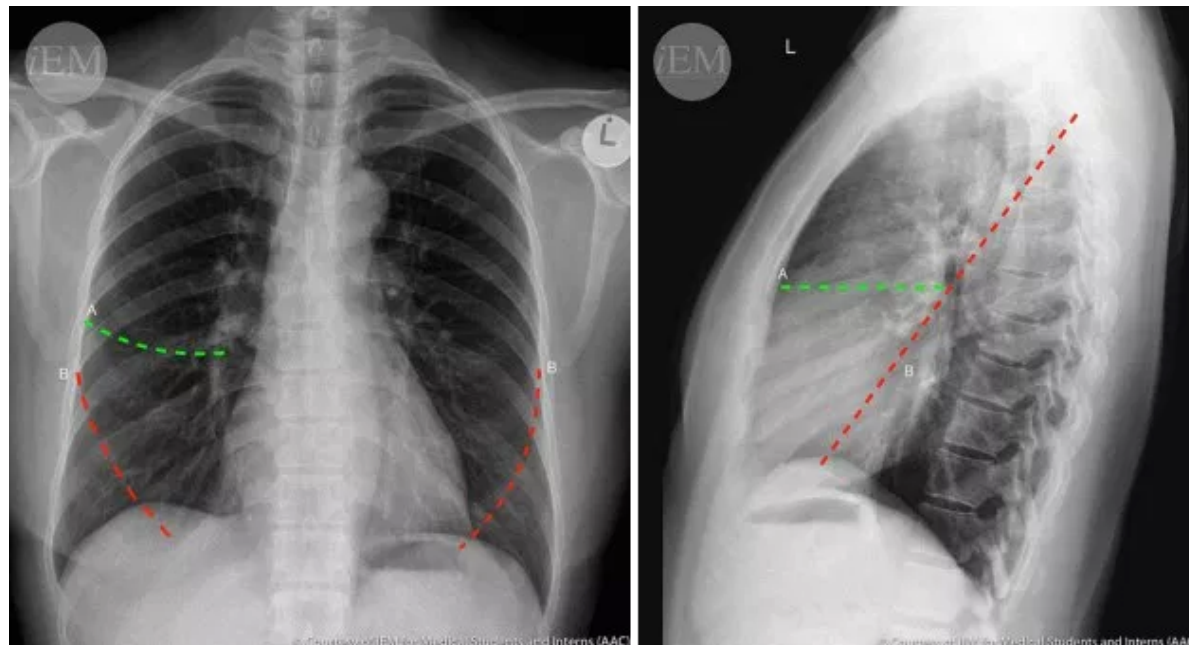
**Image 18.70** Radiological lung zones.





lines. Pay attention to the apices. You should also check for masses, consolidation, pneumothorax and vascular markings. Vessels should be almost invisible at the lung periphery. Finally, you should evaluate the major and minor fissures for fluid collection (Image 18.71).

### Image 18.71



*Minor (A) and major (B) fissures of the lung.*

Please visit our [Flickr channel](#) to see various chest x-ray pathologies.

**References and Further Reading**, click [here](#)

# How to read head CT

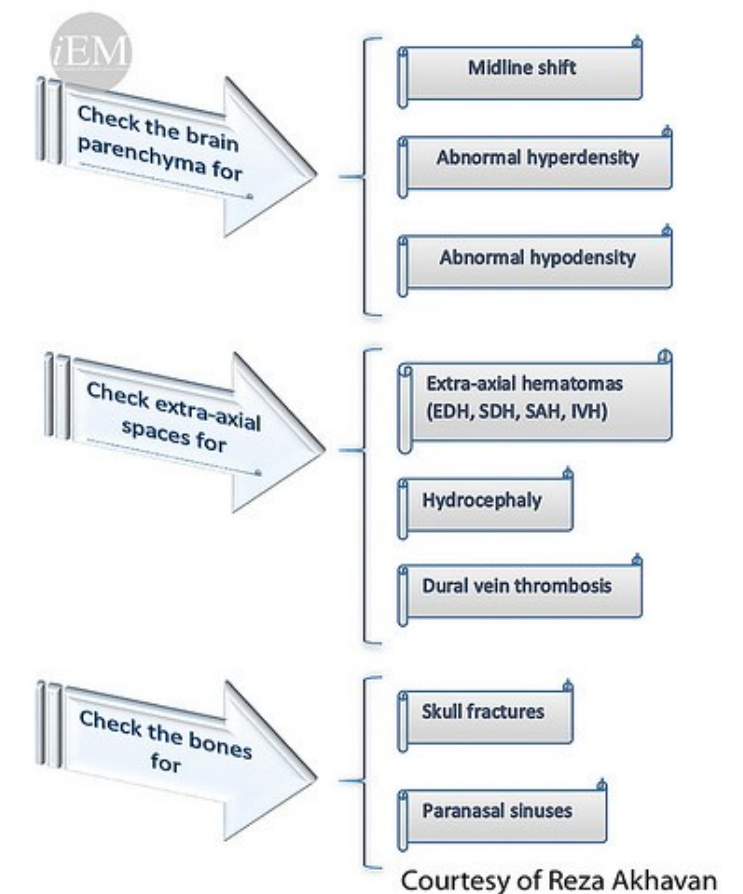
by Reza Akhavan and Bitra Abbasi

For a standard approach to read head/ brain computed tomography (CT) scan, one should adhere to systematic algorithms.

The predefined algorithms are various, and their main usage is building a mental pathway that leads the novice readers not to miss a point. Our recommended algorithm is shown in Diagram 18.1.

In the interpretation of head CT, the most critical diagnoses for emergency physicians are hemorrhage or ischemic stroke, and midline shift. These diagnoses or pathologies require immediate action. Therefore, checking the brain parenchyma is the first step for emergency physicians.

**Diagram 18.1**



*Step by step approach to brain CT in emergency situations*

# 1. Brain Parenchyma

## Midline shift

Presence of mass effect from edema or space-occupying lesions may cause a shift in midline structures. The shift of midline may cause compression on the anterior cerebral artery and eventually infarct. There are multiple sulci and cisterns in the brain that are filled with CSF. The presence of effacement in these structures is another sign for the presence of a space-occupying lesion or parenchymal edema.

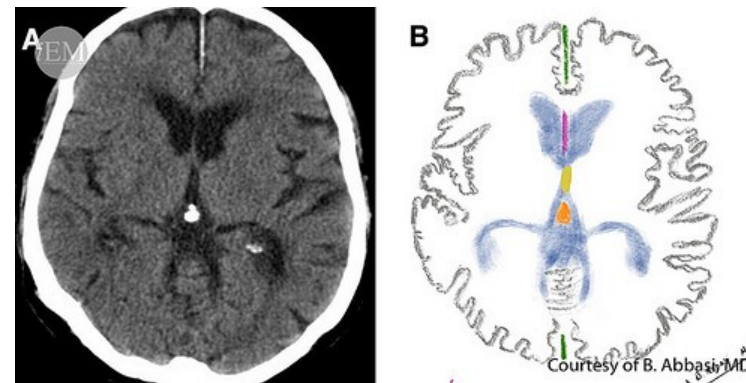
A note on anatomy (Image 18.72 and Image 18.73): There are three midline structures that should be scrutinized when searching for midline shift:

1. Septum pellucidum: a membrane located between lateral ventricles
2. Third ventricle
3. Pineal gland

All these three structures should be on the line drawn between anterior and

posterior attachments of the falx cerebri.  
\*: Bold and underlined structures are marked in accompanying figure.

**Image 18.72**

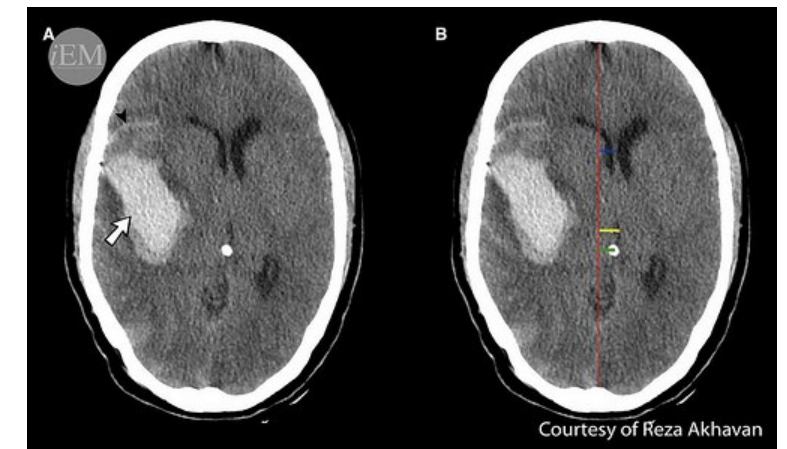


Axial brain C scan (a) and its corresponding schematic view (b) depict the midline structures. Falx cerebri (green), septum pellucidum (magenta), third ventricle (yellow) and pineal gland (orange) should be located in the midline.

## Abnormal parenchymal hyperdensity

Intraparenchymal hemorrhages manifest as hyperdense areas in brain parenchyma and are really difficult to miss! They are generally categorized as spontaneous or secondary to neoplasms, vascular malformations or trauma, etc.

**Image 18.73**



Axial non-contrast brain CT scan shows an ICH in the right parietotemporal lobe (arrow in a) with adjacent edema. SAH is seen in the brain sulci (arrowhead in a). Red-line in (b) represents the midline. Note the deviation of septum pellucidum (blue line), third ventricle (yellow line), and pineal gland (green line).

## Spontaneous hemorrhage

Also known as hemorrhagic stroke, spontaneous intracranial hemorrhages most commonly occur in hypertensive patients. The most common locations are basal ganglia, thalamus, pons, and cerebellum. Hemorrhages outside these common locations may be secondary to tumors or vascular malformation.

A note on anatomy (Image 18.74 and Image 18.75)\*: Deep grey matter nuclei

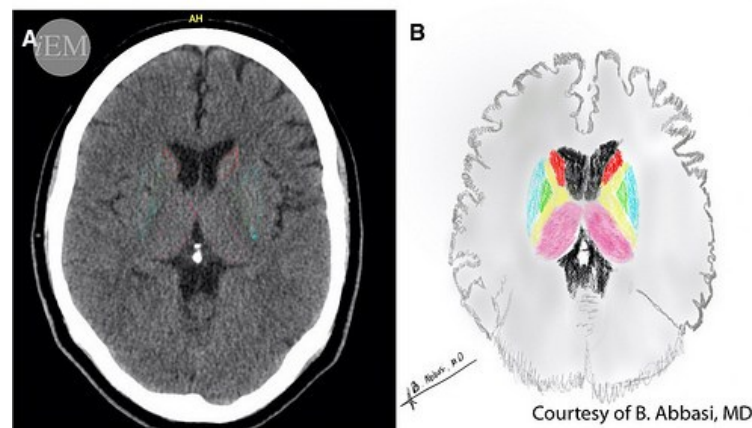


are islands of grey matter located deep in the brain:

- Thalamus
- Putamen
- Globus pallidus
- Caudate nucleus

Caudate nuclei, putamen and globus pallidus are collectively known as basal ganglia. Internal capsule is a white matter structure located adjacent to deep nuclei.

**Image 18.74**



Axial brain CT scan (a) and corresponding schematic picture (b) represent the basal ganglia and associated structures. Red: head of caudate nucleus, Green: Globus pallidus, Blue: Putamen, Pink: Thalamus, Yellow: internal capsule.

\*: Bold and underlined structures are marked in accompanying figure.

**Image 18.75**



Axial brain CT scan of a 57-year-old woman that presented with left-sided paraparesis, shows a hyperdense hemorrhage in the right basal ganglia (asterisk). Note the adjacent hypodense edema (black arrow) and mass effect on the lateral ventricle (white arrow).

### Secondary hemorrhage

The most common secondary parenchymal hemorrhages encountered

in the emergency departments are traumatic. Traumatic hemorrhages may be intra-axial (within brain parenchyma) or extra-axial. Intra-axial hemorrhages like contusions or hemorrhagic diffuse axonal injuries are discussed here. Extra-axial hemorrhages will be discussed later.

### Contusion

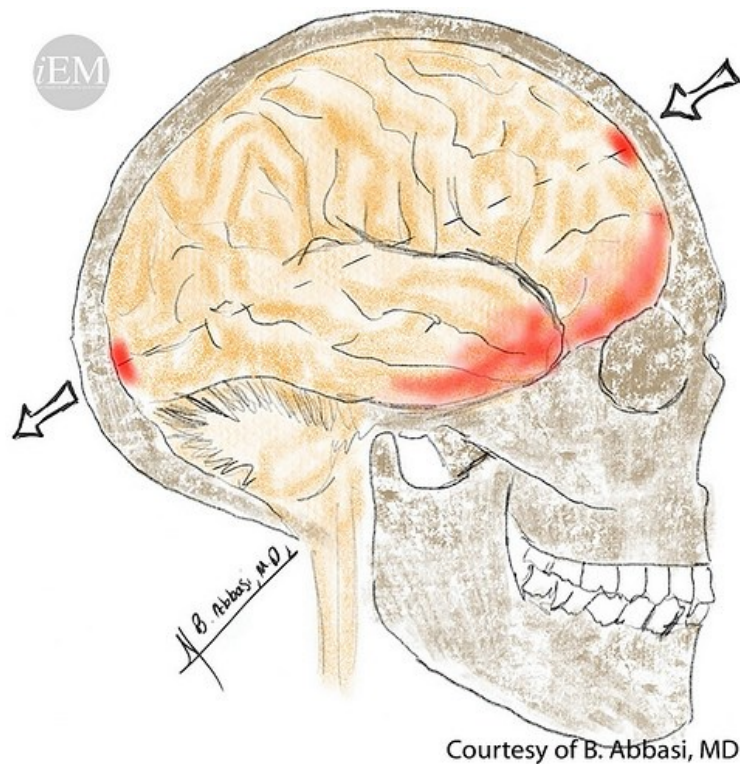
Contusions are caused by impaction of brain parenchyma on hard bony protrusions, so direct contact with bony protrusions affects cortical grey matter (Illustration 18.1). They are most commonly seen in frontal and temporal lobes (Image 18.76). They are often hemorrhagic and easily seen on computed tomography. In the control CT scans after a few days, the perilesional edema progresses, and the lesions become more readily visible.

### Diffuse axonal injury

In case of rotational acceleration traumas (brain traumas associated with rotations and change of speed), white matter and



## Illustration 18.1



Schematic representation of common locations for the contusion. Contusions most commonly occur in inferior portions of the frontal lobe, temporal lobe, underneath the direct impact (coup) or at the opposite site of direct impact (countre-coup).

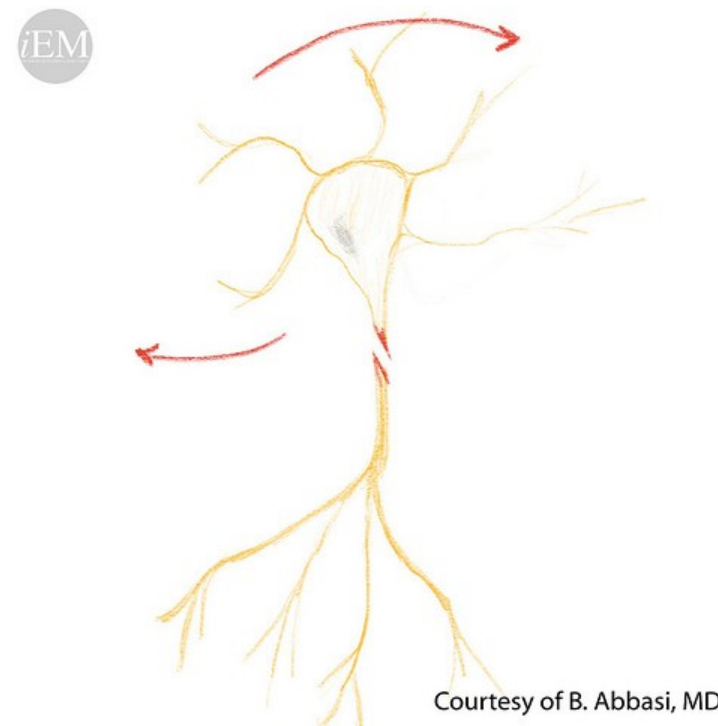
grey matter experience slightly different changes of speed. This disrupts axons at the grey-white interfaces (Illustration 18.2). This kind of lesion is called diffuse axonal injury (DAI).

## Image 18.76



Axial brain CT scan in a trauma patients shows multiple hemorrhagic lesions in the right frontal (a) and right temporal (b) lobes consistent with contusions. Note the perilesional hypodense edema.

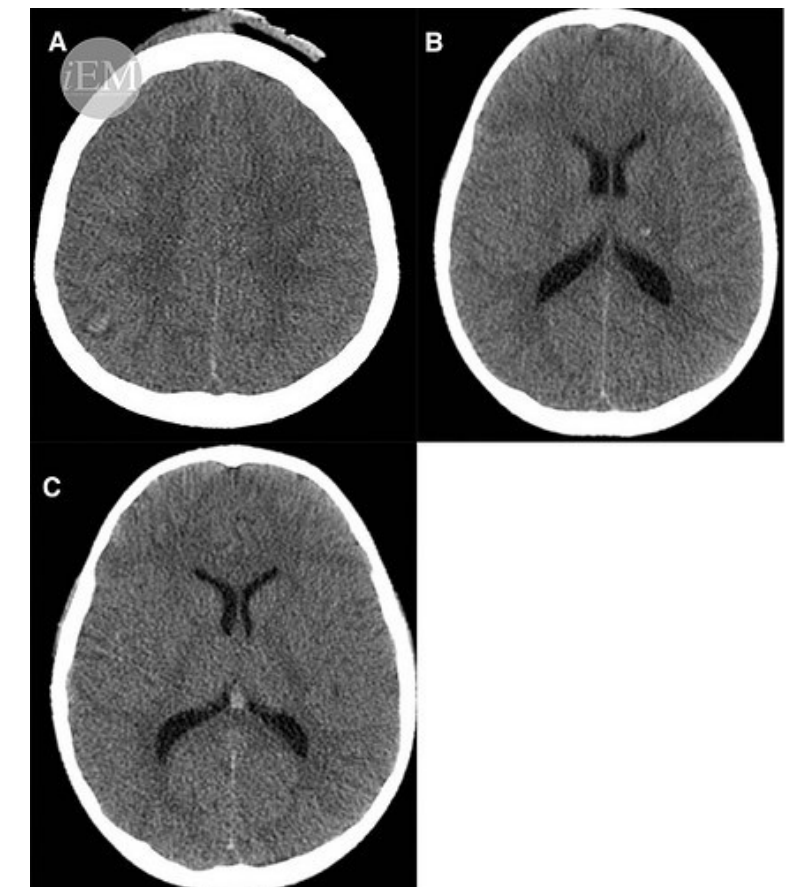
## Illustration 18.2



Schematic representation of diffuse axonal injury. Rotational forces disrupt the axons at grey-white junctions.

Unfortunately, only 15% of DAIs are visible on CT scan. MRI remains the most sensitive modality for detecting these lesions. When visible on CT, they present as hemorrhagic foci in the grey-white interface, near deep nuclei of the brain and in the corpus callosum (Image 18.77).

## Image 18.77



Axial non-contrast brain CT scan of a 34 y/o male from a motor vehicle accident. Multiple hyperdense hemorrhagic lesions are seen in the grey-white junction (a), adjacent to thalamus (b) and corpus callosum (c). Multiplicity and location of the lesions are compatible with diffuse axonal injury.

## Abnormal parenchymal hypodensity

In most cases, abnormal hypodensities represent cerebrovascular accidents (involves the cortex) or edema secondary to other pathologies (usually without cortical involvement). Stroke is defined as acute onset of focal neurologic defect due to cerebrovascular compromise. Most strokes are ischemic (80%), and some are hemorrhagic (15%). Subarachnoid hemorrhage accounts for the remaining 5%.

Brain CT scan findings in ischemic stroke are mainly a factor of time and involved artery.

In the first hours of a stroke, thrombosis in the supplying artery creates a hyperdense artery sign. This is the earliest imaging finding of acute stroke in non-contrast CT scan (Image 18.78).

Another imaging finding in non-contrast CT scan is the loss of grey-white differentiation

Image 18.78



*Axial non-contrast CT scan of a 63 y/o man with sudden onset right hemiparesis shows hyperdensity in the middle cerebral artery. This finding –being the result of arterial thrombosis– is the earliest finding in the CT scan of acute ischemic stroke. If you are able to discern the sign and diagnose a stroke in this phase, be proud of yourself!*

Cortical hypodensity (Image 18.79) and sulcal effacement (Image 18.80) are relatively late signs of stroke.

Image 18.79



*Axial non-contrast brain CT in a 49 y/o woman presented to the emergency medicine with a 3-day history of dysarthria and right-sided motor weakness. There is cortical hypodensity in the left frontal lobe (arrow) that represents ischemic infarct in the territory of the anterior cerebral artery.*



## Image 18.80



Axial non-contrast CT shows a faint hypodense area in the left frontoparietal area suggesting ischemic stroke. Sulcus effacement is noted in comparison to the other side (arrow). Note the subtlety of findings in the acute phase of stroke.

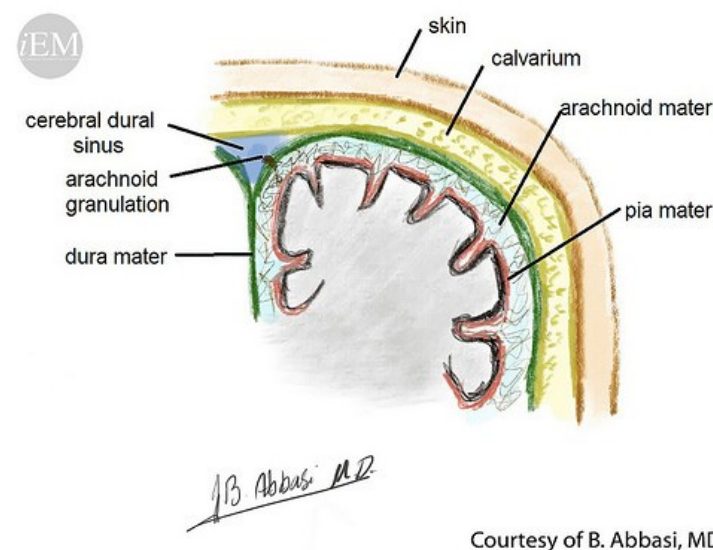
## 2. Extra-axial spaces

Extra-axial spaces are defined as the space within the skull that is not part of brain parenchyma. Meningeal layers engulf the parenchyma and separate it from the calvarium.

The meninges are composed of three layers: pia mater (that is in direct contact

with grey matter and contains supplying capillaries), arachnoid network (that contains CSF and absorbs it into dural veins via arachnoid granulations) and dura mater (that is in direct contact with periosteum). The layers are depicted schematically in Illustration 18.3.

### Illustration 18.3



Schematic representation of meningeal layers.

The most common extra-axial pathologies are traumatic.

### Epidural hematoma

Epidural space is a potential space located between the periosteum and dura mater. These layers have a tight

connection, and it takes a considerable amount of force for them to be separated. In practice, epidural hematomas are mostly due to arterial hemorrhage (especially middle meningeal artery) and are usually associated with skull fractures (Illustration 18.4).

### Illustration 18.4



Schematic representation of epidural hematoma. Note that epidural hematomas do not cross suture lines and may be associated with skull fractures.

Epidural hematomas appear as biconvex or lentiform hyperdensity in the brain periphery (Image 18.81). They may cross the midline but do not cross the sutures. This is because of the attachment of dura to the sutures.

**Image 18.81**



Axial non-contrast brain CT scan shows a lentiform epidural hematoma in the right hemisphere.

**hematoma**

Subdural space is a potential space between the inner layer of dura and arachnoid membrane (Illustration 18.5). Hematomas within subdural space are usually due to rupture of bridging veins located in this areas.

Subdural hematomas appear as crescent-shaped collections of blood overlying the cerebral

## Subdural

**Illustration 18.5**



Schematic representation of subdural hematoma. Note that subdural hematomas may cross the suture lines, but do not cross the midline.

**Image 18.82**



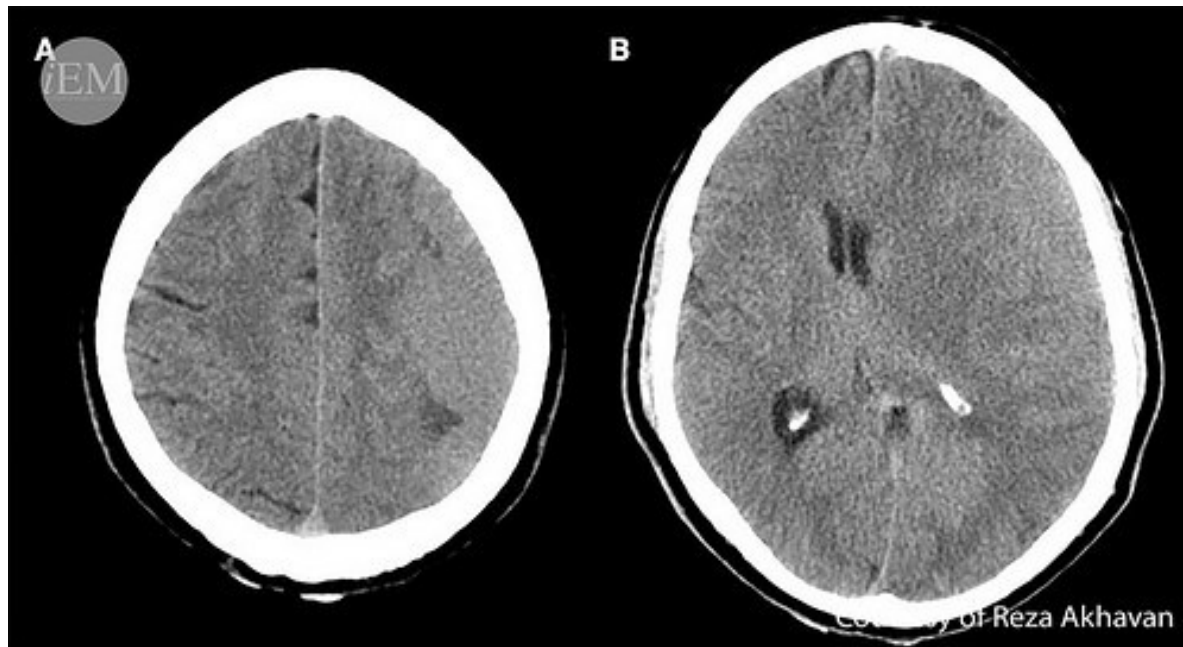
axial non-contrast brain CT scan shows an acute SDH in the left hemisphere with midline shift.

hemispheres (Image 18.82). They may cross the sutures or track along the falx or tentorium, but do not cross the midline.



As time progresses, the density of SDH decreases. Therefore subacute SDH is isodense to brain parenchyma and becomes gradually hypodense in chronic stages. In the subacute phase, SDH may not be readily discernible, and attention to the midline shift helps in diagnosis (Image 18.83).

**Image 18.83**



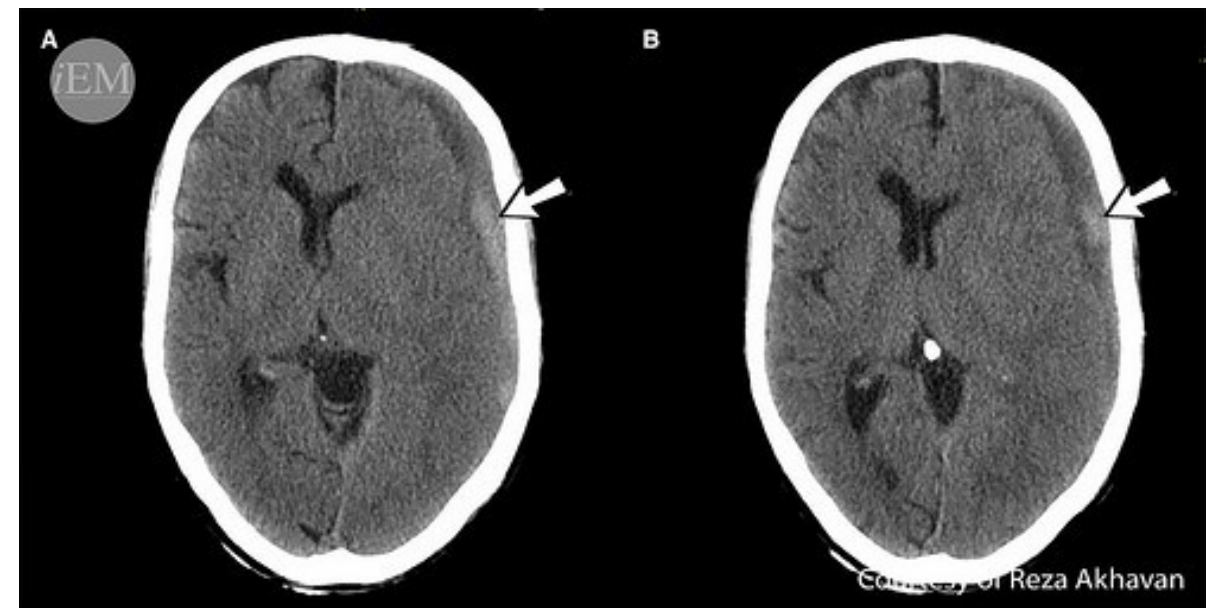
Axial brain CT-scan reveals a midline shift. As a novice image interpreter, you may see no other pathologies. Look carefully at the left hemisphere! What appears as a thickened cortex, is actually an isodense subacute SDH, hence known as thick cortex sign.

The layered appearance of SDH is a sign of active rebleeding. Call the neurosurgeon immediately (Image 18.84)!

## Subarachnoid hemorrhage (SAH)

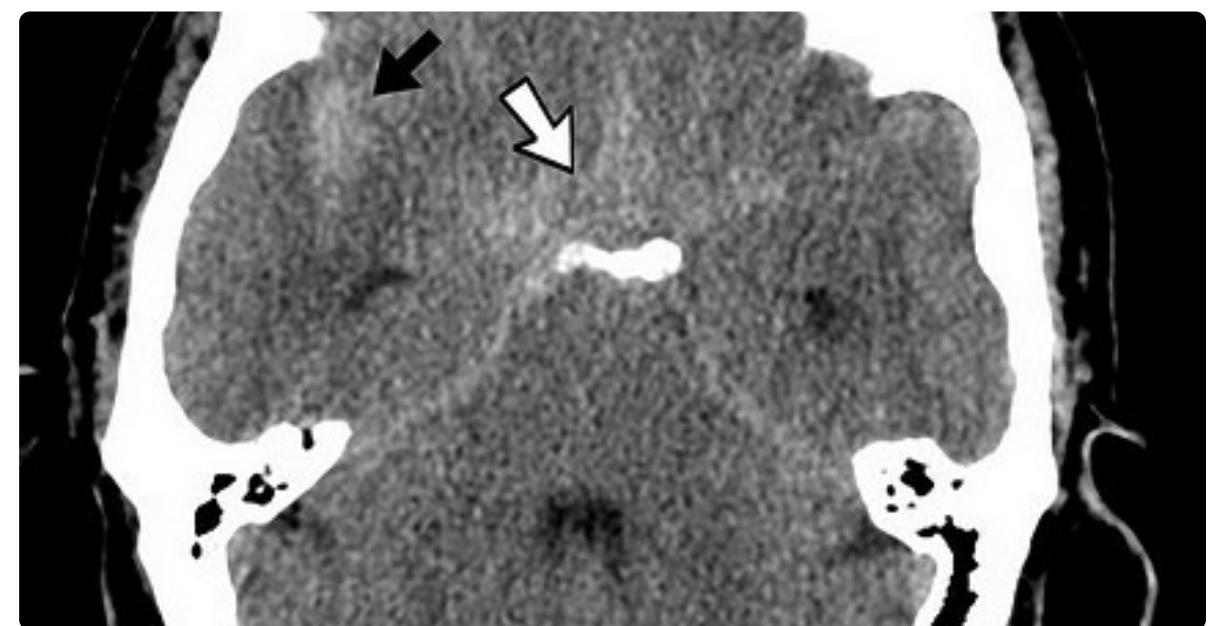
Subarachnoid space is the space between subarachnoid membrane and pia mater. The space contains CSF and extends

**Image 18.84**



Axial non-contrast brain CT scan shows the chronic phase of epidural hematoma. There are areas of increased density (arrows in a and b) in the EDH that are compatible with active rebleeding.

**Image 18.85**



This patient presented to the ED complaining of a severe headache. Axial non-contrast brain CT-scan shows hyperdense SAH in the basal cisterns (white arrow) and right-sided Sylvian fissure (black arrow). Note the hypodensity in the adjacent parenchyma that represents secondary edema.

into brain sulci and cisterns. In the CT scan, SAH is seen only in the acute phase and presents as hyperdensity within the sulci and cisterns (Image 18.85).

Subarachnoid hemorrhages may be traumatic or non-traumatic. Traumatic SAH is usually associated with SDH. Non-traumatic SAH is usually due to a ruptured berry aneurysm.

### Dural vein thrombosis

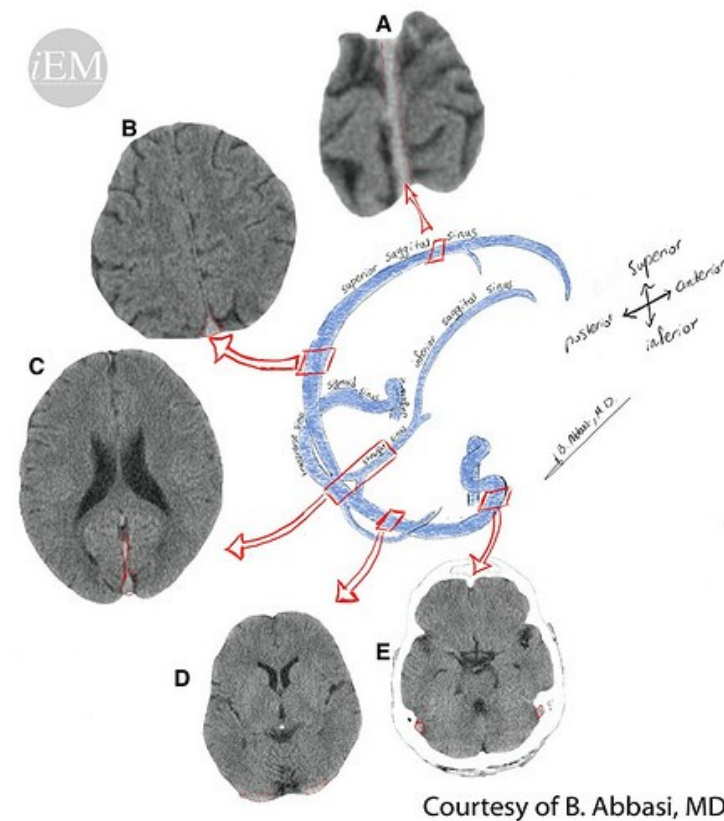
Dural veins are located between dural layers. Increased density in these structures is a sign of dural vein thrombosis.

A note on anatomy (Illustration 18.6 and Image 18.86)\*: Dural veins are venous structures of the brain that are located between dural layers. In addition of draining venous blood, they have the critical task of CSF resorption. Dural vein thrombosis causes increased pressure in the venous structures and may lead to infarcts and hemorrhages. This may also cause increased intracranial pressure by

hindering CSF resorption.

\*: Bold and underlined structures are marked in accompanying figure.

### Illustration 18.6



A schematic view of main dural veins and their correspondence on axial brain CT scans. Superior sagittal sinus (a and b), straight sinus (c), transverse sinuses (d) and sigmoid sinuses (e) are outlined on the CT images.

### Intraventricular hemorrhage (IVH)

The most common cause of IVH in adults is trauma. Blood in the ventricles could

### Image 18.86

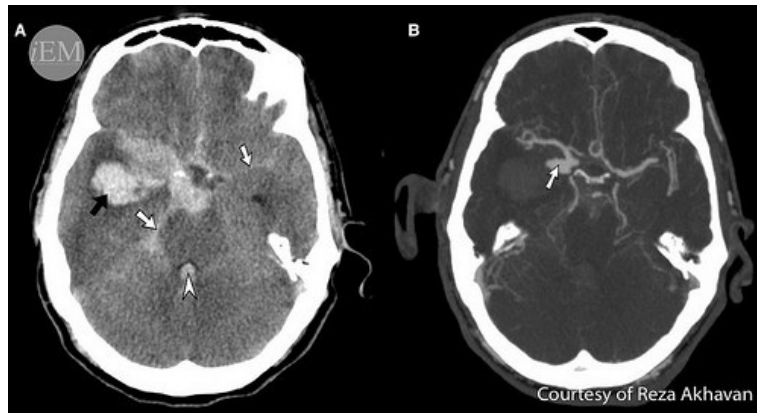


Axial non-contrast brain CT scan shows increased density in the posterior part of superior sagittal sinus indicating thrombosis (arrowhead). There is also a hyperdense intracranial hematoma (ICH) in the left parietal lobe (arrow) with adjacent edema. Remember that intraparenchymal hematoma is due to impairment of blood flow and venous infarct secondary to dural vein thrombosis.

enter the structure from adjacent parenchymal hemorrhage or blood in the subarachnoid space (SAH) may enter the ventricle via CSF flow (Image 18.87).



**Image 18.87**



A 55 y/o man presented with sudden loss of consciousness. Axial non-contrast brain CT scan (a) shows ICH in the right temporal lobe (black arrow), SAH in basal cisterns (white arrows in a) and IVH in the fourth ventricle (arrowhead). As the ICH was located outside the common locations of spontaneous ICH, a CT angiography of cerebral vessels was performed (b). In CT angiography an outpouching of contrast was noted (white arrow in b) that is consistent with an aneurysm.

## Hydrocephalus

Hydrocephaly or dilatation of ventricles (Image 18.88) may be the result of mass-occupying lesion that obstructs CSF flow in ventricular structures or may be due to impairment of CSF resorption via subarachnoid granulations.

**Image 18.88**



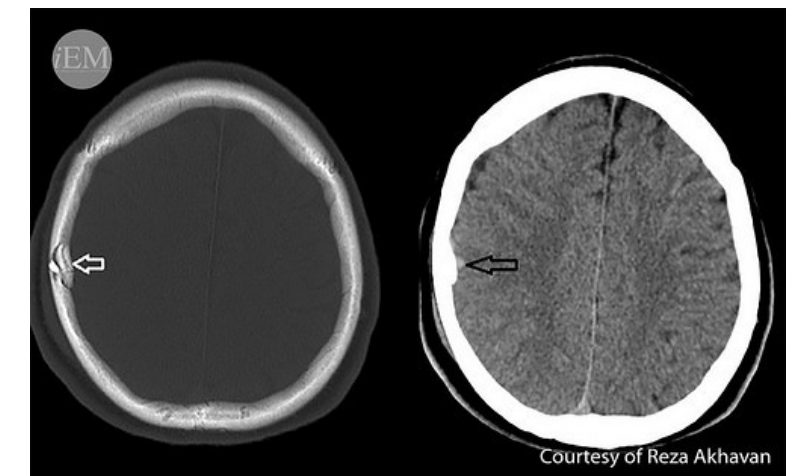
Axial non-contrast brain CT scan shows dilatation of lateral ventricles that suggests hydrocephaly.

## 3. Bones

The presence of skull fractures is not always a sign of underlying brain injury. However, the significant amount of force that causes a skull fracture mandates careful evaluation of other associated intracranial injuries.

Simple linear fractures are the most common types of skull fractures and appear as linear hypodense lines in the skull. Depressed skull fractures are usually accompanied with contusions in the underneath brain parenchyma (Image 18.89).

**Image 18.89**

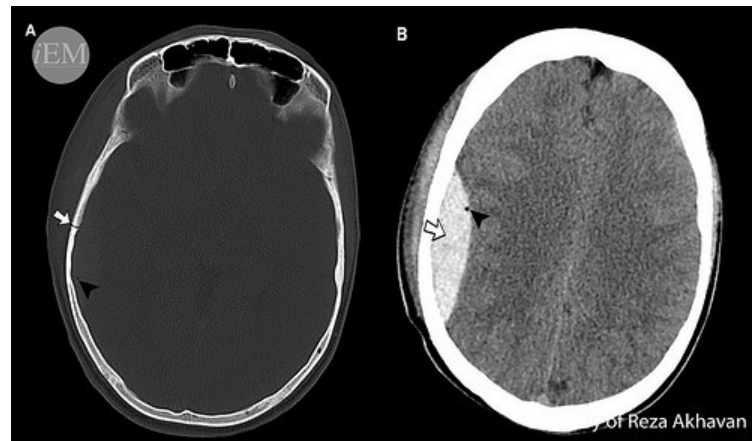


Axial brain CT-scan in a young man with a shotgun injury. Bone window (a) reveals a depressed skull fracture (arrow in a). Note the hyperdense pellet embedded near the fracture. Parenchymal window (b) shows a contusion (arrow in b) in the adjacent parenchyma.

Warning signs in a skull fracture include the presence of intracranial air (pneumocephalus)(figure 28), depression of inner table of calvarium (depressed fracture), overlying scalp laceration (open

skull fracture), or fractures adjacent to dural veins or middle meningeal artery (figure 28). Whenever you encounter a skull fracture, look carefully at these red flags!

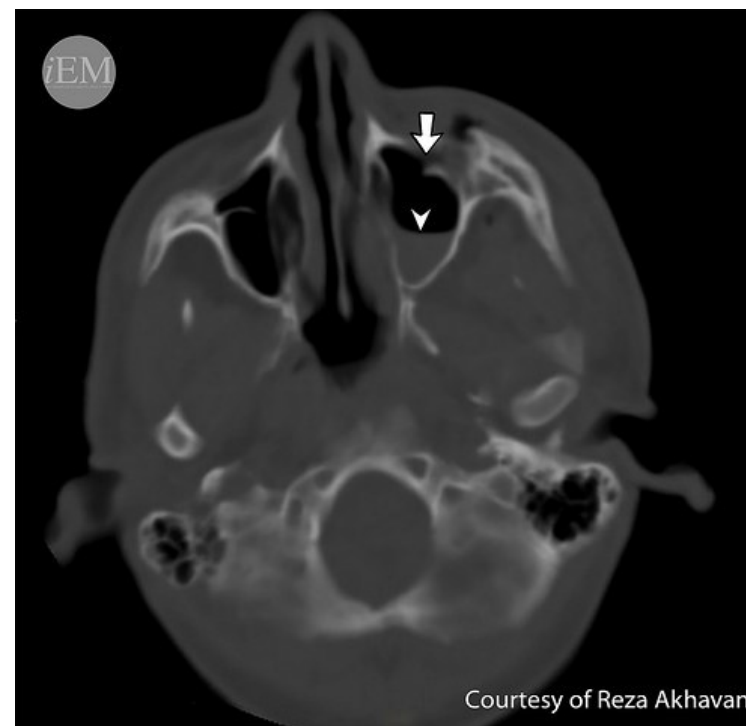
**Image 18.90**



Axial non-contrast brain CT scan in a trauma patient. Bone window (a) shows a linear skull fracture (arrow in a). The location of the fracture near middle meningeal artery and presence of pneumocephalus (arrowheads in a and b) warrants further evaluation. Parenchymal window (b) depicts an epidural hematoma (arrow in b) that is secondary to the rupture of the middle meningeal artery.

Finally, note the visible parts of paranasal sinuses and look for possible fractures. The presence of air-fluid levels or hyperdensity within the sinuses might be an indication of fracture in the trauma setting (Image 18.91)

**Image 18.91**

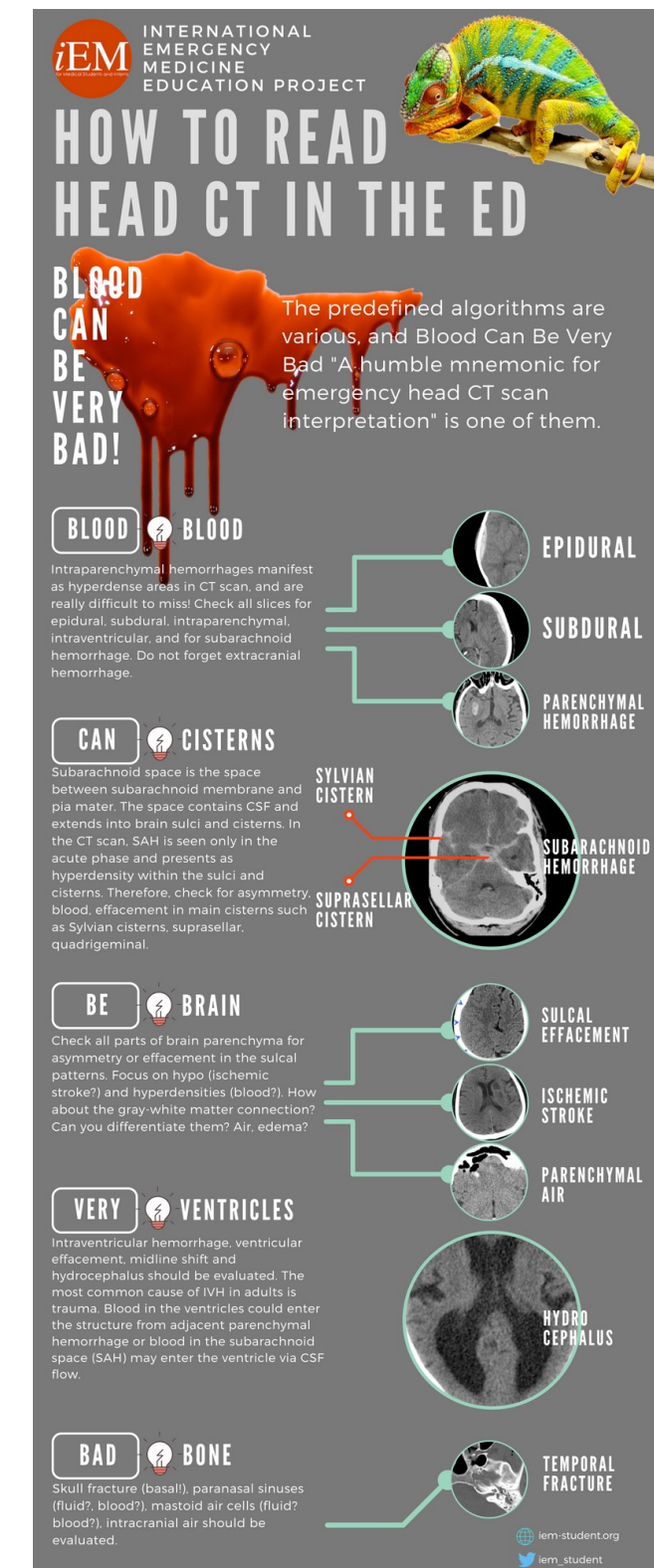


Axial non-contrast bone window CT scan of the brain shows a fracture in the anterior wall of the left maxillary sinus (arrow). The air-fluid level within the sinus (arrowhead) indicates hemorrhage.

Another CT head interpretation mnemonic is BLOOD CAN BE VERY BAD. We will not go over all details of this mnemonic, but we have an infographic for you (Illustration 18.7). Please visit our [Flickr channel](#) for more pathologic CT scan images.

**References and Further Reading**, click [here](#)

**Illustration 18.7** How to read CT





# How to read pelvic x-rays

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by Sara Nikolić and Gregor Prosen

## Case Presentation

*A 27-year-old woman was in a car accident. She is hemodynamically stable with vital signs as follows: temperature of 36.4°C, heart rate of 70 bpm, blood pressure 120/80 mmHg, respiratory rate 10/min, oxygen saturation 99% on room air. During the secondary survey, pelvic bones are not stable, and there is a pain on palpation. You placed a pelvic binder and ordered a pelvic X-ray.*

## Introduction

Pelvic fractures carry life-threatening injury potential which should be identified or suspect during the primary assessment of patients with major trauma. The prevalence of pelvic fracture in studies of patients with blunt trauma is between 5% and 11.9%. The mortality from pelvic fractures in patients who reach hospital is reported to be between 7.6% and 19%. Usually, injuries are secondary to massive force, such as a road traffic accident or fall from a height. Fractures may be associated with vascular, soft tissue and visceral injuries. If the pelvic ring is broken in two places, the fracture is likely to be unstable. Isolated ring fractures, however, tend to be stable. Patients who survive a pelvic fracture are at risk for

significant complications such as chronic pain, leg length discrepancy, sexual dysfunction, or nerve palsy.

## Important Anatomical Considerations

The three bones compose the pelvis (the sacrum and the two innominate bones). Strong ligaments keep these three bones together. These are crucial for maintaining pelvic stability. A large array of ligaments traverses the interior and exterior surface of the posterior aspect of the pelvis. Two ligaments originate from the side and back of the sacrum and insert into the ischial spine and ischial tuberosity.

The pubic symphysis, a fibrocartilagenous joint, is supported by ligaments. However, adds little to the overall stability of the pelvis. The urethra and bladder lie close to the pubic symphysis, and there is a 20% risk of injury if symphysis is disrupted.

Torn or rupture of the ligaments can cause separation of three bones. In this situation, the nerves and vessels running

close to them, especially at the posterior, can also be injured. The bleeding is usually venous and extraperitoneal and can be life-threatening.

If bones fracture but the ligaments remain intact, a tamponade effect can be achieved, and the degree of hemorrhage limited.

## Mechanism of Injury

The Young-Burgess system identifies four types of pelvic ring disruption, based on interpretation of radiographic images: anteroposterior compression, lateral compression, vertical shear and combined mechanical injury.

**Anteroposterior compression** causes “open book” look at one or both sides of the pelvis. A diffuse force will disrupt the pubic symphysis, while a more direct force fractures the pubic rami in a vertical plane. For the pubic bones to separate by over 2,5 cm, one or both of the ligaments associated with sacroiliac joints have to be torn. An anteroposterior force can also push the flexed femur backward so that

the femoral head impacts and fractures the posterior margin of the acetabular rim.

**Lateral compression** produces a horizontal fracture through the ipsilateral pubic symphysis and momentary medial displacement of the hemipelvis. A lateral compression force can also impinge on the upper femur causing central dislocation of the hip.

**Vertical shear forces** the hemi-pelvis upwards and towards the midline and can tear all the sacroiliac ligaments on the affected side as well as the pubic symphysis ligaments.

**Complex pattern** happens in less than 25% of cases. The pelvis is exposed to two or more of the forces mentioned above. A combination of injuries results in a complex radiological picture.

## X-Ray Views

The routine pelvic view is anteroposterior (AP) projection, and in 94% of cases, a correct diagnosis can be made from this

view. When the fracture is noted in the AP view, special views (inlet and outlet view and oblique views) for further investigations are recommended.

**Radiographic interpretation is systematized with ABCS approach:**

- Alignment
- Bones
- Cartilage and joints
- Soft Tissues

**Image 18.92**



## Normal findings

### AP View Interpretation Summary

#### A

##### • Adequacy and quality

Ensure that the whole of the pelvis is visible

##### • Alignment

Assess the borders of the three circles namely, the pelvic brim and the two obturator foramina.

#### B

##### • Bones: Check each of the following systematically:

- Pubis Sacrum
- Acetabulum
- Femoral heads
- Iliac crest
- Lumbar vertebrae

#### C

##### • Cartilage and joints

##### • Check the pubic symphysis

- Check the sacroiliac joints
- Check the acetabulum

#### S

##### Soft Tissues

- Check the disruption of fat planes inside the pelvis
- Check for soft tissue shadows outside the pelvis

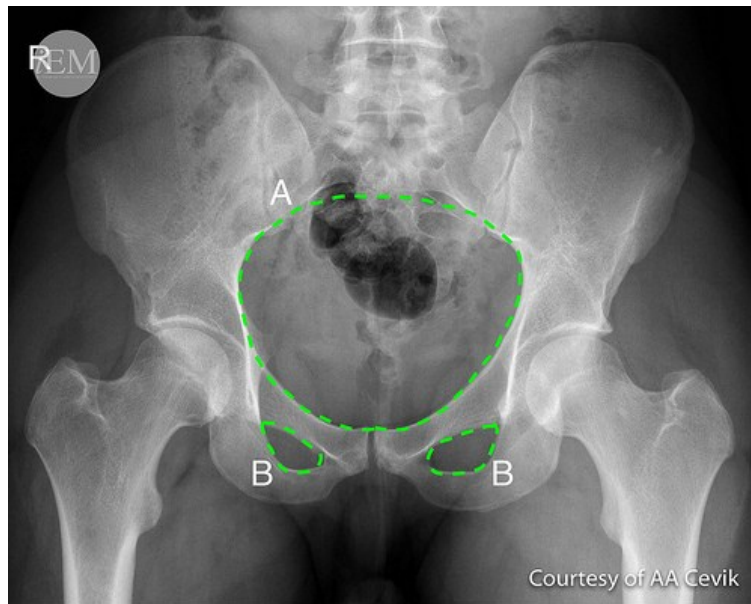
## Details

#### A

In this step, focus the three circles enclosed by the pelvis. One is created by the pelvic brim (A) and the other two by the obturator foramina (B) (Image 18.93).

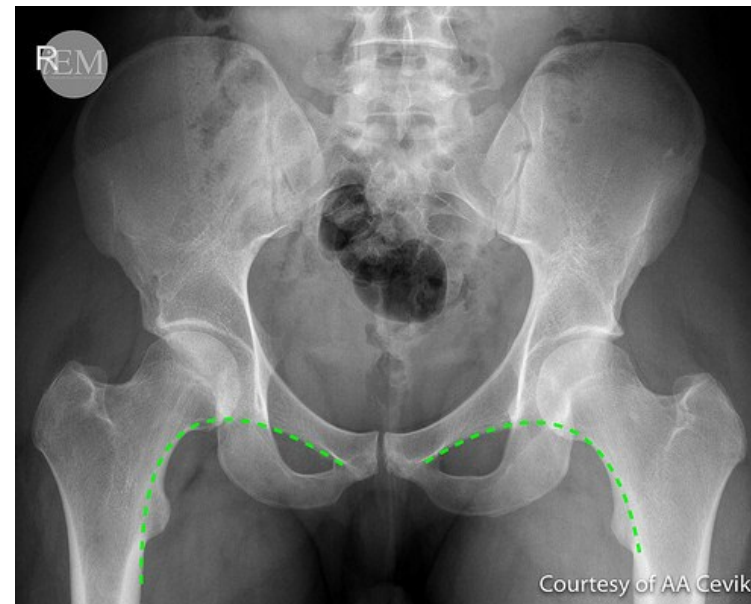
Trace around the edge of the large circle. Normally this has a smooth edge which is not disrupted by the sacroiliac joint or pubic symphysis unless the patient is very old. The pelvic brim cannot be disrupted in only one place. As the pelvis is not completely rigid, this disruption

**Image 18.93**



may take the form of a minimal diastasis. The inner margins of both obturator foramina should then be inspected in the same way as the pelvic brim. Again these are rarely broken in only one place. Complete the examination of the foramina by tracing along its superior border to the inferior surface of the neck of the femur. This is known as Shenton's line (Image 18.94).

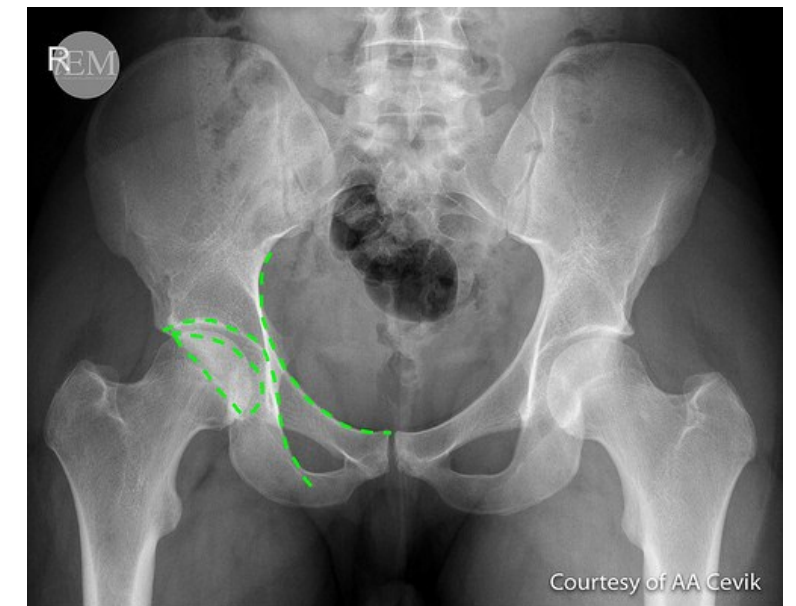
**Image 18.94**



**B** Examine the outer edges of the pelvis and its bony structure for evidence of fractures. These may present as areas of increased density, lucency, or alteration of the internal trabecular pattern. Fractures away from the three bony circles can occur in isolation. The start point of the examination is the pubic symphysis. Then, slowly progress to the right or left side. Focus on the posterior and anterior joint margin, the ilioischial line (posterior column), and the iliopectineal line (anterior column). To finish the exam, look

for "teardrop sign" (acetabular floor) (Image 18.95 and 18.96).

**Image 18.95**



Next, focus on the anterior inferior iliac spine, anterior superior iliac spine and look for the iliac crest to the sacrum. The sacrum should also be examined for symmetry of its foramina (Image 18.97).

**C** Check for either widening or overlapping of bones at the level of the symphysis pubis (A). If you see one of those, disruption in the pelvic brim should be investigated. Sacroiliac joints (B) at the right and left sides must also be checked



Image 18.96

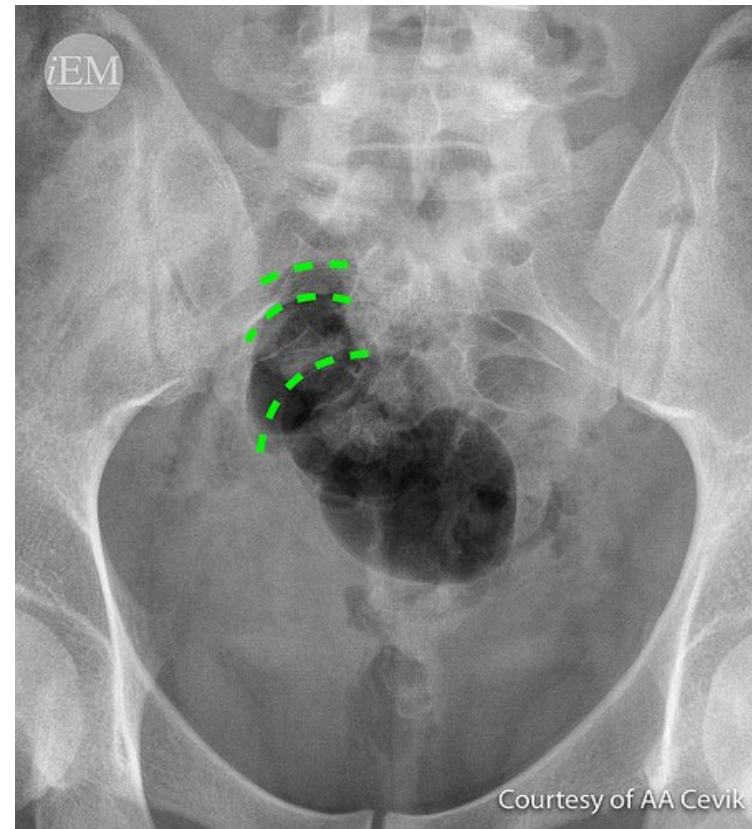


for widening, defects in the cortical surface, overlapping of bone, and lack of congruity of the joint margin (Image 18.98).

## S

Check for soft tissue shadowing both inside and outside the pelvis because hematoma and tissue edema can produce swellings which are visible on the anteroposterior radiograph. Normally the obturator internus muscle is seen on both sides of the pelvis as a dark grey line, which is due to the muscle or fat

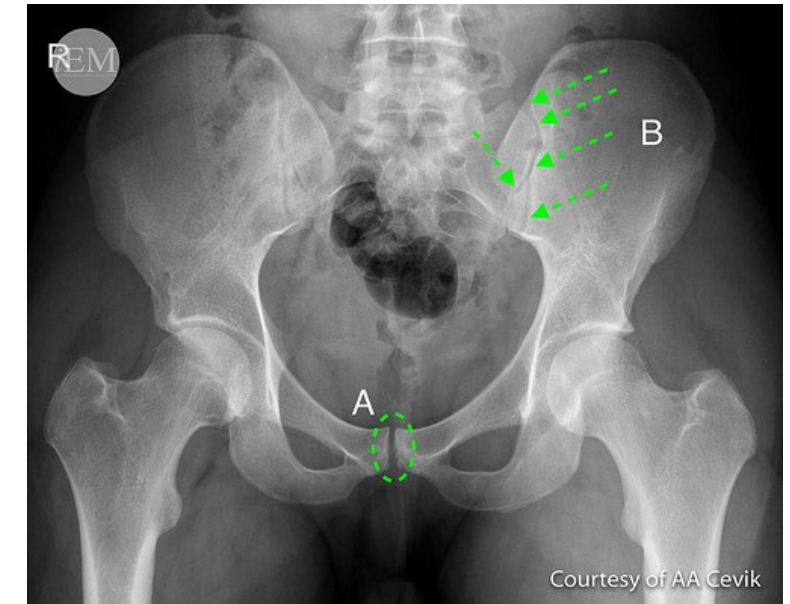
Image 18.97



plane. Loss of this line indicates extra-peritoneal hemorrhage or soft tissue edema. Conversely, intra-peritoneal hemorrhage can displace the line.

Inlet and outlet views should ideally be requested if there is clinical or radiological evidence of a pelvic fracture. An inlet view looks down the lumen of the true pelvis. It is better than the anteroposterior view for showing the orientation of fractures of the pubic rami.

Image 18.98



Outlet views are used to detect the degree of vertical displacement of the fracture fragments.

Oblique (Judet) views are used to define acetabular fracture patterns. If a fracture or abnormality of the acetabulum is suspected computed tomography will usually be necessary once the patient has been adequately resuscitated and stabilized.



## Abnormal Findings

**Image 18.99** Pubic rami fracture



**Image 18.101** Complex pelvic fracture – open book fracture.



*Pay attention to symphysis pubis and sacroiliac joint separations.*

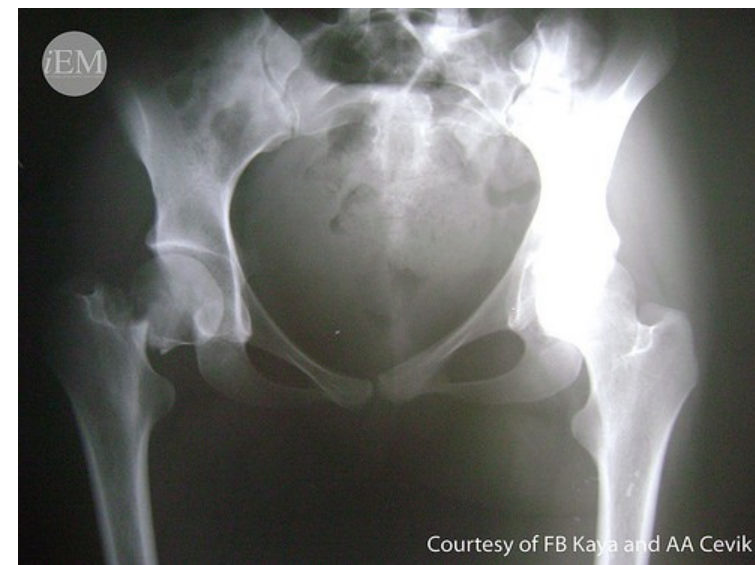
**Image 18.104** Hip dislocation (antero-inferior)



**Image 18.100** Pubic rami and ischium fracture



**Image 18.102** Femoral neck fracture



*Right femoral neck fracture after fall. Check the Shenton's line alignment.*

**Image 18.103** Hip dislocation (posterior)



**Image 18.105** Acetabular fracture



## Hints and Pitfalls

Ensure that the whole of the pelvis can be seen, including the iliac crests, both hips, and the femurs distal to the lesser trochanters. The adequacy of the penetration should also be assessed. Pelvic rotation is determined by lining up the symphysis pubis with the midline of the sacrum.

It is common for part of the iliac crest to be missing or poorly penetrated in the films so that fractures cannot be seen. A rotated film causes asymmetry of the bony circles and the sacroiliac

joints. Failing to trace around the bony edges, especially the iliac crests and sacral foramina, will lead to fractures being missed.

Epiphyseal lines may be misinterpreted as fractures. Remember that the Y-shaped (triradiate) cartilage separating the pubis, ischium, and ilium in the acetabular floor does not fuse until puberty.

Accessory ossification centers (in particular the one in the posterior acetabulum) may also be mistaken for fractures. However, apophyses are usually bilateral, have a sclerotic margin, and are not associated with overlying soft tissue signs.

Being systematic is crucial to make possible for the non-specialist to interpret pelvic radiographs accurately. Table 1 shows the summary of how to read a pelvic x-ray.

**References and Further Reading**, click [here](#)



## Chapter 19

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# Selected Emergency Drugs





# Antidotes

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by Hamidreza Reihani and Elham Pishbin

An antidote is an agent or drug that can reverse the toxic effects of poisoning. The base of clinical practice in the treatment of toxicities is cardiopulmonary stabilization, decontamination, enhancing elimination and supportive management. Antidotes are indicated in some specific and well-defined situations, and they are not routinely administered in toxin exposures. Therefore, the physician should know the indications and contraindications of each antidote. Administration of the pharmacologic antagonists may worsen the outcome in some situations and are not recommended.

There is usually enough time to start the treatment after supportive care and evaluation of the patient. However,

there are a few indications that antidotes should be administered as soon as possible to prevent major complications and death. Cyanide antidotes for cyanide toxicity, naloxone for severe opium overdose and atropine for organophosphate poisoning and gas agents are some examples.

The antidotes administered in clinical practice are not a great deal. There is a list of essential antidotes and a brief explanation of their character and application.

## Antidotes and their characteristics

### Atropine

#### General information

- Anticholinergic agent
- Competitive muscarinic antagonist

#### Indications

- Organophosphate poisoning
- Carbamates
- Nerve agents

#### Precautions

- Excessive doses lead to anticholinergic symptoms

#### Dose

- Start with 1-2 mg IV (adults), 0.02 mg/kg IV (children)
- Double the dose every 2-3 minute to achieve atropinization

#### Administration

- Infusion of 10-20% of stabilizing dose/ hour
- Large doses may be required
- There are auto-injectors for rapid use

#### Other

- Drying of respiratory secretions is the goal
- Tachycardia is not the endpoint

### Calcium

#### General information

- Calcium chloride 10% (1 g /10 mL), (27.2 mg/mL elemental Ca)
- Calcium gluconate 10% (9 mg/mL elemental Ca), one-third of the calcium in strength of calcium chloride

#### Indications

- Calcium channel blockers toxicity
- Hydrofluoric acid exposure
- Hyperkalemia

- Hypermagnesaemia

#### Precautions

- Calcium chloride extravasation can lead to soft tissue necrosis, preferably administered via central line
- Continuous monitoring is recommended

#### Dose

- 1 gram calcium chloride (10 mL), (0.15mL/kg in children)
- 10-30 mL of calcium gluconate

#### Administration

- IV bolus over 5 minutes
- Repeated doses every 10-20 minutes if needed
- Infusion can be administered

#### Other

- Topical calcium gel or local injection of calcium gluconate for hydrofluoric acid skin burns

- Intra-arterial or IV with a Bier block for extremity exposure

## Ciproheptadine

### General information

- An antihistaminic and antiserotonergic agent
- Has anticholinergic effects as well

### Indications

- Control of symptoms in serotonin syndrome

### Precautions

- May cause anticholinergic effects

### Dose

- 8 mg (adults), 4 mg (children) not approved

### Administration

- Oral
- Can be repeated every 8 hours until 24 hours

## Deferoxamin

### General information

- Iron-chelating agent
- Converts it to a water-soluble complex excreted by urine

### Indications

- Systemic iron toxicity
- Iron levels > 500 µg/dL
- Multiple pills on radiography

### Precautions

- Hypotension may occur at rapid rates

- Cardiac monitoring is needed

- Avoid infusion more than 24 hours

### Dose

- Start with 15 mg/kg/h

### Administration

- IV infusion
- Infusion rate could be increased

### Other

- Evaluate patient after 6 hours
- The urine color will become red

## Digoxin immune Fab

### General information

- Fab fragments of antibodies to digoxin

- Reverse the dangerous cardiac effects of digitalis

### Indications

- Acute and chronic digoxin overdose
- Other cardiac glycosides poisoning

### Precautions

- Close monitoring; ready for resuscitation
- Monitor serum free level of digoxin

### Dose

- Acute overdose: 5 vials; for unstable patients 10-20 vials; can be calculated if the ingested dose is known
- Chronic overdose: can be calculated by serum digoxin level; start with 2 vials

## Administration

- Bolus in life threatening conditions, otherwise infusion

## Other

- For other cardiac glycoside poisoning start with 5 vials

## Dimercaprol

(BAL)

## General information

- Heavy metal chelator

## Indications

- Severe lead, inorganic arsenic and mercury poisoning

## Precautions

- Many severe adverse effects
- Nephrotoxic
- Administered in ICU

## Dose

- 3 mg/kg

## Administration

- IM every 4 hours for 48 hours

## Ethanol

## General information

- Blocks the formation of toxic metabolites of alcohols

## Indications

- Methanol and ethylene glycol poisoning

## Precautions

- Serum ethanol levels monitored every 1-2 hours
- The dose should be doubled during dialysis

## Dose/ Administration

- IV: loading (10 mL/kg of 10% IV solution), maintenance (1-2 mL/kg/h of 10% IV solution)

- Oral: loading (1.5-2 mL/kg 80-proof liquor), maintenance (0.2-0.5 mL/kg/h)

## Other

- Maintain blood ethanol concentration between 100-150 mg/dl

## Flumazenil

## General information

- Competitive benzodiazepine antagonist
- Not used routinely in benzodiazepine poisoning

## Indications

- Reversal of procedural sedation
- Pediatric poisoning (limited use)

## Precautions

- May cause withdrawal
- May induce seizure

## Dose

- 0.2 mg, 0.01 mg/kg (children)



- Repeat up to the desired effect or 3 mg

### Administration

- IV over 30 seconds

### Other

- Not used in mixed drug overdose

## Fomepizole

### General information

- An alcohol dehydrogenase inhibitor

### Indications

- Methanol and ethylene glycol toxicity

### Dose

- Loading dose = 15 mg/kg
- Maintenance dose = 10 mg/kg q12 hours

### Administration

- Infusion in 100 ml normal saline or 5% dextrose in 30 minutes

### Other

- In dialyzed patients is given every 4 hours or continuous infusion

## Glucagon

### General information

- Increase cAMP\*\*
- Positive inotropic and chronotropic similar to beta-agonists

### Indications

- $\beta$ -blocker toxicity
- Calcium channel blocker toxicity

### Precautions

- Induces vomiting, consider airway management

### Dose

- 5-10 mg (adults), 0.05-0.1 mg/kg (children)

### Administration

- The first dose is IV bolus, if there is a clinical response, start infusion

## Hydroxocobalamin

### General information

- A precursor of Vitamin B12
- Hydroxyl group is displaced by cyanide and form cyanocobalamin

### Indications

- Cyanide toxicity

### Precautions

- It's a safe drug

### Dose

- 5 g, repeat if needed; 70 mg/kg (children)

### Administration

- Infusion in 100 normal saline in 15 minutes

### Other

- Skin and urine orange-red discoloration

## Insulin (High dose)

### General information

- It has strong inotropic effects

### Indications

- Calcium channel blocker toxicity
- Beta-blocker toxicity

### Precautions

- Glucose level should be monitored every 10 minutes
- Hypokalemia be considered

### Dose/ Administration

- 1 IU/kg IV bolus of short acting insulin, followed by 0.5-1 IU/kg/hr
- Glucose 25 g (dextrose 50%) before starting insulin, then 25 g/hr according to glucose level

### Other

- Higher doses were administered in studies

## Intravenous Lipid Emulsion

### General information

- 20% lipid emulsion as a parenteral nutrient

### Indications

- Overdose by drugs with high protein binding and large volume of distribution, e.g. Local anesthetics,  $\beta$ -blockers and calcium channel blockers

### Dose/Administration

- 1.5 ml/kg IV bolus
- 0.25 ml/kg/minute

### Other

- Until hemodynamic stability restored

## Methylen blue

### General information

- It reduces methemoglobin (MetHb) to hemoglobin

### Indications

- Methemoglobin-forming agents toxicity
- Symptomatic methemoglobinemia
- MetHb levels >20% in asymptomatic patients

### Precautions

- Pulse oximetry is unreliable in methemoglobinemia
- Hemolysis in G6PD deficiency

### Dose

- 1-2 mg/kg IV, 1 mg/kg (children)

### Administration

- Slow IV injection, may repeat 30-60 minutes later

### Other

- MetHb levels measured frequently

## N-acetylcysteine

### General information

- Preventing hepatocellular injury in severe acetaminophen toxicity

### Indications

- Serum acetaminophen concentration above toxic level
- Hepatocellular injury

### Precautions

- Oral therapy may not be tolerated due to its taste and odor

### Dose/Administration

- Oral: loading (140 mg/kg), then (70 mg/kg q 4 hours) for 17 doses
- IV: loading 150 mg/kg in 30-60 minutes then 50 mg/kg over 4 hours, 100 mg/kg infused over next 16 hours

## Naloxone

### General information

- An opioid antagonist

### Indications

- For reversing the opioid effects, respiratory and CNS depression

### Precautions

- Re-sedation may occur due to short half-life of naloxone
- Withdrawal in chronic users

### Dose

- Start: 0.1 to 0.4 mg; 0.01 mg/kg (children)
- Repeat every 2-3 minutes up to 10 mg

### Administration

- Intravenously, intramuscularly, or subcutaneously

### Other

- Start with larger doses if respiratory depression exist
- Infusions may be required

## Octreotide

### General information

- Synthetic analogue of somatostatin

### Indications

- Hypoglycemia due to sulfonylurea

### Precautions

- Break through hypoglycemia may occur

### Dose/Administration

- 50 µg IV then 25 µg/h or
- 100 µg IM or SC every 6 hours

## Physostigmine

### General information

- Reverse anticholinergic syndrome

### Indications

- For CNS symptoms (delirium, seizure) due to anticholinergic drugs

### Precautions

- Contraindicated in bradycardia, AV block and bronchospasm

### Dose

- 0.5 – 1 mg (adults), 0.02 mg/kg (children)

### Administration

- IV slowly in 2 minutes or IM
- Repeat in 10 to 30 minutes if needed

### Other

- In rapid administration cholinergic symptoms may occur

## Pralidoxime

### General information

- Reactivate cholinesterase inhibition due to organophosphates

### Indications

- Organophosphates poisoning
- Nerve agents

### Dose

- Loading: 1-2 g IV; 25-50 mg/kg (children)

- Maintenance: 500 mg/hr or 1-2 g q4-6h; 10-20 mg/kg/hour (children)

### Administration

- IV infusion in 0.9% saline

### Other

- Should be administered in the early phase before irreversible binding occurs

## Pyridoxine

### General information

- Vitamin B6 is essential for GABA production

### Indications

- Isoniazid, hydrazine and Gyromitra poisoning
- Ethylene glycol poisoning

### Dose/Administration

- 1 gram for each gram of isoniazid, 70 mg/kg (children), maximum 5 gram

- 50 mg IV every 6 hours for ethylene glycol toxicity

- 0.5 g/min infusion until the seizure stops

## Sodium bicarbonate

### General information

- Hyperosmolar Sodium Bicarbonate Injection

### Indications

- Cardiotoxicity due to fast sodium channel blockade (e.g., TCA\* poisoning)
- Urine alkalinization

### Precautions

- Hypokalemia is a concern
- Serum pH maintained between 7.50-7.55

### Dose

- Start with 1-2 mEq/kg, further doses may be needed



## Administration

- First dose administered bolus
- Other bolus doses or infusion if required

## Other

- Given only if there is evidence of cardiotoxicity, such as QRS widening and ventricular dysrhythmias

## Sodium calcium edetate (EDTA)

### General information

- IV heavy metal chelator

### Indications

- Severe lead toxicity
- Lead level > 70 µg/dl

### Precautions

- Patient should be admitted in hospital
- Nephrotoxicity, ECG changes and liver test disturbance may occur

### Dose

- 25-75 mg/kg/day

## Administration

- Continuous infusion for 5 days

## Other

- Usually starts 4 hours after first dimercaprol (BAL) injection

## Sodium thiosulfate

### General information

- Help the body to detoxify cyanide

### Indications

- Cyanide poisoning

### Precautions

- In severe cases with other antidotes

### Dose

- 50 ml of 25% (12.5 g; 1 ampoule) in adults; 1.65 ml/kg (children)

### Administration

- IV over 10 minutes

- Repeat after 30 minutes if clinically needed

## Succimer (DMSA)

### General information

- Oral metal chelator

### Indications

- Symptomatic lead poisoning
- Asymptomatic lead poisoning, lead level > 60 µg/dl (adults), > 45 µg/dl (children)

### Precautions

- May cause neutropenia, gastrointestinal upset and liver abnormalities

### Dose

- 10 mg/kg three times a day for 1 week, then two times a day for 2 weeks

### Administration

- Orally

### Other

- The serum level should be monitored

**References and Further Reading**, click [here](#)

# Drugs for Pain Relief

---

by Nik Ahmad Shaiffudin Nik Him, Azizul Fadzi

## Introduction

A patient presented with pain at the Emergency Department (ED) commonly un-recognized, under-treated and delayed in getting treatment. Prompt recognition and alleviation of pain should be a priority when treating patient suffered from pain. There are many drugs available for pain relief. Optimal control of pain is essential for good patient care. It prevents an adverse physiological and psychological effects, reduce the incidence of chronic pain, postoperative morbidity and facilitate earlier discharge from the hospital.

**Drugs for pain relief may be used for:**

1. Acute pain

2. Chronic pain

## Acute versus Chronic Pain

Pain is an unpleasant feeling. Sensory neurons convey it to the brain by as a result of injury, disease, or emotional disorder. Acute pain is defined as pain less than 6 months duration with a known cause and disappears when the problem resolves. Chronic pain occurs when pain lasts more than 6 months duration, persists beyond the healing time and usually the cause may not be determined (International Association for the study of pain, 2007).

The scientific approach to pain management demands a step-wise approach, which utilizes lower risk interventions first (WHO, 1996) especially in acute pain management. It is important to understand the

different pain mechanisms of chronic pain as well as evidence-based multi-mechanistic treatment. It is also essential to provide individualized treatment. Pharmacological and non-pharmacological aspect is as equally important in chronic pain management at decreasing pain and increasing functioning of chronic pain patients during activity of daily livings.

The classes of medications used in the treatment of pain (Adapted from ACPA resource guide to chronic pain medication & treatment, 2015) include:

1. Non-opioids (simple, non-selective and selective COX-2 inhibitors) e.g. aspirin, NSAIDs, and acetaminophen and celecoxib.
2. Opioids (weak and strong) e.g., tramadol, morphine, codeine, hydrocodone, and oxycodone.
3. Adjuvant analgesics e.g. antidepressants, anticonvulsants
4. Local analgesia, e.g. lidocaine patch

5. Others: Medications with no direct pain-relieving properties may also be prescribed as part of a pain management plan e.g. laxative, anti-emetic, steroids, bisphosphonates, muscle relaxant and anti-spasmodic

The tables below shows some specific information about these agents.

**Table 19.1** Non-opioids (Simple analgesic)

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Paracetamol	0.5 - 1gm, 6 - 8 hourly Max: 4g/day Reduce maximum dose 50%-70% in patients with hepatic impairment	Rare	Hepatic impairment	Preferred drug in elderly.  Liver damage following over dosage.  Maximum dose 4 g daily.
Perfalgan (IV) Aqueous solution: 10mg/ml paracetamol, available in 50ml and 100ml vials	>50 kg, 1 g 6 hourly up to max 4g/day 10-50 kg, 15 mg/kg/dose max 60mg/kg in 4 divided doses  Administration: Infusion over 15 minutes. Renal & hepatic impairment: minimum interval between doses should not be less than 6 hours		Hepatic impairment	Important to consider the total dosage of paracetamol used i.e. to include dosage of suppositories and oral preparations.

*Provided by authors*



**Table 19.2** Non-Opioid (Non-Selective NSAIDs)

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Aspirin	325 to 650 mg orally or rectally every 4 hours as needed, not to exceed 4 g/day.	Peptic ulcer, GI bleed, Platelet dysfunction, Renal failure, Hypertension Allergic reaction in susceptible individuals, Increase in CVS events  Same for below agents	Gastroduodenal ulcer Asthma Bleeding disorder Renal dysfunction Ischaemic heart disease Cerebrovascular disease Inflammatory bowel disease  Same for below agents	Current data suggest that increased CVS risk may be an effect of the NSAIDs/Coxib class.  Physicians and patients should weigh the benefits and risks of NSAIDs/Coxib therapy.  Concurrent use with aspirin inhibits aspirin's antiplatelet effect (mechanism unclear)  Same for below agents
Diclofenac Sodium	50 - 150 mg daily, 8 - 12 hourly Max: 200 mg/day			
Mefenemic Acid	250-500 mg 8 hourly			
Ibuprofen	200-400 mg, 8 hourly Max: 2400 mg/day Elderly patients: 200 mg 3 x a day			
Naproxen	500-550mg BD Elderly patients; 220 mg BD			
Ketoprofen	Patch: 30 -60 mg BD Topical; PRN			
Ketorolac	IV: 10-20 mg BD ( max 3days)			
Meloxicam	7.5-15 mg daily Max: 15 mg /day			

*Provided by authors*

**Table 19.4** Non-Opioids ( Selective Cox-2 Inhibitors)

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Celecoxib	400mg BD in acute pain (48 hours only) 200-400 mg daily (for longer term use) <18 years : not recommended Elderly patients: 100 mg daily	Renal impairment Allergy reaction in susceptible individuals Increase in CVS events Hypertension  Same for below agents	Ischaemic heart disease Cerebrovascular disease Hypersensitivity to sulfonamides. Higher doses associated with higher incidence of GIT, CVS side effects. Patients with indications for cardioprotection require aspirin supplement Uncontrolled Hypertension  Same for below agents	Associated with lower risk of serious upper gastrointestinal side effects compared to traditional NSAIDs  Use the lowest effective dose for the shortest duration necessary
Etoricoxib	120 mg daily in acute pain (48 hours only) 60 - 90 mg daily (for longer term use) Elderly patients 30 mg daily			
Parecoxib	20-40mg 6-12 hourly (max 80mg/day for max duration of 48 hours ) Elderly (>65 years & <50kg) reduce to half the dose with a maximum daily dose of 40mg.  Renal & hepatic impairment : Do not use			

Provide by authors

**Table 19.3** Opioids (Weak opioids)

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Tramadol	50 - 100 mg, 6 - 8 hourly Max: 400 mg/day	Dizziness Nausea Vomitting Constipation Drowsiness	Risk of seizures in patients with history of seizures and with high doses In elderly, start at lowest dose (50 mg) and maximum 300 mg daily	Interaction with TCA, SSRI and SNRI
Dihydrocodeine tartrate (DF118)	30 - 60 mg, 6 - 8 hourly Max: 240 mg/day  Renal dysfunction & dialysis patient: do not use  Hepatic dysfunction: do not use	Nausea Vomiting Constipation Drowsiness	Respiratory depression Acute alcoholism Paralytic ileus Raised intracranial pressure	Metabolites can accumulate causing adverse effects  In severe hepatic impairment, codeine may not be converted to the active metabolite- morphine.

Provided by authors

**Table 19.5** Combinations of opioids and paracetamol

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Paracetamol 500 mg + Codeine 8 mg	1 - 2 tablets, 6 - 8 hourly Max: 8 tablets/day	Constipation	Hepatic impairment	Decrease in side effect profile of Codein/ tramadol respectively and paracetamol while maintaining efficacy
Paracetamol 325 mg + Tramadol 37.5 mg	1 - 2 tablets, 6 - 8 hourly Max: 8 tablets/day	Nausea Vomiting Drowsiness	Hepatic impairment, Epilepsy	Same as above

*Provided by authors*

**Table 19.6** Opioids ( Strong opioids)

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Morphine	<p>SC (Adults): &lt;65 yrs: 5mg-10 mg 4 hrly &gt;65 yrs: 2.5 mg-5mg 4hrly</p> <p>IV: Follow morphine pain protocol (Appendix)</p> <p>Oral: Starting dose 5- 10 mg, 4 hourly of IR</p> <p>Elderly: 2.5 - 5 mg, 4 - 6 hourly of IR</p>	<p>Nausea Vomiting Pruritus Sedation Constipation Respiratory depression Myoclonus</p>	<p>Acute bronchial asthma</p> <p>Respiratory depression</p> <p>Head injuries, Renal and hepatic dysfunction: needs dose adjustment</p>	<p>Metabolites can accumulate causing increased therapeutic and adverse effects</p> <p>Both parent drug and metabolites can be removed with dialysis, watch for “rebound” pain effect</p>
Fentanyl	<p>To be prescribed by APS team only</p> <p>Renal dysfunction : appears safe, however, a dose reduction is necessary</p> <p>Dialysis patients : appears safe</p> <p>Hepatic dysfunction : appears safe, generally no dose adjustment necessary</p>	<p>Nausea Vomiting Sedation Constipation Respiratory depression</p>		<p>No active metabolites and appears to have no added risk of adverse effects; monitor with high long term user</p> <p>Metabolites are inactive, but use caution because fentanyl is poorly dialysable</p> <p>Decrease hepatic blood flow affects metabolism more than hepatic failure.</p>
Oxycodone IR (oxynorm)	<p>Starting dose (oral): 5 -10 mg 4 - 6 hourly</p> <p>Renal dysfunction : Use cautiously with careful monitoring, adjust dose if necessary</p> <p>Dialysis patients: do not use</p> <p>Hepatic dysfunction: Use cautiously and monitor patient carefully for symptoms of opioid overdose Decrease initial dose by 1/2 to 1/3 of the usual amount Elderly patients : 2.5-5 mg every 4-6 h</p>	<p>Nausea Vomiting Sedation Constipation Respiratory depression</p>	<p>Acute bronchial asthma Respiratory depression Con-comittent used of sedative drugs Head injuries, Renal and hepatic dysfunction: needs dose adjustment</p>	<p>Metabolites and parent drug can accumulate causing toxic and CNS-depressant effects</p> <p>In severe hepatic impairment, the parent drug may not be readily converted to metabolites</p>

*Provided by authors*



**Table 19.7** Adjuvant Therapies

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Antidepressant				
Amitriptyline	Start with 10 - 25 mg nocte. Increase weekly by 25 mg/day to a max of 150 mg/day  Elderly patients: 10 mg ON	Anticholinergic effects e.g. dry mouth, drowsiness, urinary retention, arrhythmias	Not recommended in elderly patients with cardiac disease, glaucoma, renal disease	Nortriptyline may be a suitable alternative and better tolerated in elderly at similar doses  Interaction with Tramadol Significant risk of adverse effects for the elderly
Duloxetine	30 - 60 mg/day Max: 120 mg/day	Gastrointestinal disorder Excessive sweating CNS disorder	Narrow-angle glaucoma Potent CYP1A2 inhibitors Concomitant use of MAOIs Hypertension	Interaction with Tramadol
Anticonvulsants				
Carbamazepine	100 - 1600 mg/day  Elderly patients: 100 mg daily	Dizziness Ataxia Fatigue Leucopenia Nausea Vomiting Drowsiness	Increased ocular pressure Latent psychosis Confusion Agitation	Well tolerated. Serious adverse events are rare
Gabapentin	Day 1: start at 300mg Day 2: 300 mg 12 hourly Day 3: 300 mg 8 hourly Thereafter, increase by 300 mg/day every 1- 7 days Max: 3600 mg/day  Elderly patients : 100mg daily	Drowsiness dizziness GI symptoms Mild peripheral oedema	Dose adjustment needed in renal impairment	However, need to monitor sedation, ataxia, oedema, hepatic trans-aminases, blood count , serum creatinine, blood urea and electrolytes
Pregabalin	Start with 150 mg/day (in 2 divided doses). If needed, increase to 300 mg/day after 3 - 7 days intervals, then if needed, increase to 600 mg/day after 7 days interval Max: 600 mg/day Elderly patients : 50 mg at bedtime	Same as above	Same as above	Same as above

*Provided by authors*

**Table 19.8** Other agents used for analgesia or an adjunct to analgesics

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Bisphosphonates				
Pamidronate	60 - 90 mg as a single infusion over 2 - 4 hrs every 4 weeks	Asymptomatic hypocalcemia, hypophosphataemia, hypomagnesaemia Flu-like symptoms Mild fever Local injection -site reactions Malaise Rigor	Hypersensitivity to biphosphonates.  Hyperparathyroidism  In renal impairment, reduce dose and increase infusion duration required  In patients with poor dental hygiene, there is higher risk of ONJ. Dental referral is advised	Rehydrate patients with normal saline before or during treatment.  Not to be given as bolus injection
Zoledronate Acid	4 mg as 15 min IV infusion every 3 - 4 weeks	Hypertermia Flu-like symptoms Headache Hypersensitivity Osteonecrosis of jaw	Same as above	Same as above
Steroids as anti inflammatory				
Dexamethasone	Oral/ IV/SC: 8 - 16 mg daily or divided doses (initial dose), then to reduce to lowest possible dose (usually 2 mg/day)  Elderly patients :5 mg daily and taper as soon as feasible	Increased or decreased appetite Insomnia, Indigestion, Nervousness Myopathy, Oral candidiasis Adrenal suppression	Peptic ulcer disease Concomitant NSAIDs use Liver or cardiac impairment	Should be given before 6 pm to reduce risk of insomnia  Efficacy may reduce over 2 - 4 weeks Use lowest possible dose to prevent side effects.  Anticipate fluid retention and glycemic effects in short-term use and CV and bone demineralization with long-term use Monitor for rash or skin irritation
Lignocaine (topical)				
Lignocaine 5%	Elderly patients : 1-3 patches for 12 hours per day			Monitor muscle weakness, urinary function, cognitive effects, sedation
Muscle relaxant				
Baclofen	5 mg -15 mg daily			Avoid abrupt discontinuation because of CNS irritability
Laxatives				

**Table 19.9** Other agents used for analgesia or an adjunct to analgesics

DRUG	RECOMMENDED DOSAGES	SIDE EFFECTS	CAUTIONS AND CONTRAINDICATIONS	COMMENTS
Lactulose	15 - 45 ml orally 6 - 8 hourly	Bloating, Epigastric pain Flatulence, Nausea, Vomiting Cramping	Hypersensitivity to lactulose products Galactosemia  Patients requiring a galactose free diet	May be mixed with fruit juice, water or milk Reasonable fluid intake is required for efficacy
Bisacodyl	5 - 10 mg orally, 1 - 2 times daily Max: 30 mg/day	Atony of colon	Intestinal obstruction	
Antiemetic				
Metoclopramide	10 - 20 mg 6 - 8 hourly	Extrapyramidal reactions Dizziness Drowsiness	Epileptic patients Gastrointestinal hemorrhage	
Haloperidol	0.5-3 mg ON	Extrapyramidal Syndromes Dystonia Prolonged QT interval Neuroleptic Malignant Syndrome	Concomitant use with other psychotropic drugs may increase Extra-pyramidal Syndromes	
Granisetron	1 mg 12 hourly	Constipation	Progressive ileus and/or gastric distension may be masked	Should not be used as first line. Not for long term use.
Ondansetron	8 mg 12 hourly	Headache Sensation of flushing or warmth in the head and epigastrium Constipation	Pregnancy and lactation Hepatic impairment	
Prochlorperazine	10 - 30 mg daily in divided doses Severe nausea and vomiting: 20 mg stat followed by 10 mg after 2 hours For prevention: 5 - 10 mg 8 - 12 hourly	Extrapyramidal symptoms Dry mouth	May increased risk of seizure with Tramadol	

Provided by authors

## Management of Major Opioid Complications

### Hypoventilation\* or Unarousable

1. Stop infusion
2. Oxygen 12L/min via Hudson mask
3. Naloxone (Narcan) 0.01mg/kg  
\*Hypoventilation if
  - Respiratory rate < 10 / min. for > 5 years old
  - Respiratory rate < 15 / min. for 1 – 5 years old
  - Respiratory rate < 20 / min. for < 1 year old.

### Apnoea

1. Stop infusion
2. Ventilate with bag and mask (100% oxygen)
3. Check pulse, if absent start CPR
4. Naloxone (Narcan) 0.01mg/kg

## Severe Vomiting

1. Before any antiemetic, always ensure that patient is adequately hydrated, good analgesia, and that hypoglycemia and hypotension are not causative factors.
2. Reduce or stop infusion if necessary.
3. Give Ondansetron 0.15mg/kg IV or Granisetron 0.05mg/kg IV over 10 min.

## Hints and Pitfalls

### The hints

1. More than 75% of ED presenting complain is related to pain.
2. Severe pain creates a barrier to obtain an adequate history and physical exam. It can be easily resolved by giving early pain medication thus facilitate better patient care.
3. Assessment of pain severity is challenging that requires a holistic approach. Thus, regardless of one's preferred approach, the assessment method should be used and

supplemented with regular pain reassessments.

4. Drugs for pain relief should be chosen appropriately and to keep it simple as polypharmacy is associated with more side effects.
5. Pain medication should be given within 20–25 minutes of initial evaluation at ED including the plan of treatment. The benefits not only improving patient comfort but also had physiological advantages, e.g. reduction of pain-related tachycardia in acute coronary syndrome and aortic dissection.
6. History of medication that had been taken and failed prior to ED presentation is crucial. It should be known that medications which have failed at home are likely to fail in the ED.
7. Always consider targeted analgesia.
  - No doubt non-specific analgesics (e.g., NSAIDs, opioids) useful in the ED but the risk of side effects



may be significant in certain population thus targeted analgesia is the best approach.

- ED providers should consider a specific and effective therapy available, e.g. local nerve block

8. In acute severe pain, fast administration of drugs for analgesia is better and preferable.

- The key with regard to analgesia administration route is neither “always use IV” and nor “the more severe the pain, the more likely IV is the right route.”
- In difficult IV access or IV route’s disadvantages seem to outweigh its benefits, alternative approaches may be best

9. Any suspicions of drug abuse, e.g. preference or insist on certain opioids, ECP should obtain a detailed history and consider for referral to psychologist for evaluation for drug abuse

10. Pain care is an ongoing process in the ED and after discharge.

- Ignorance of the principle of ongoing pain treatment lead to risks of “wind-up” and increased analgesia requirements
- Proper pain care saves time overall (as for a fracture), it will likely be necessary for at least a few days and often more after discharge

### The pitfalls

1. The response to drugs for pain relief varies for individual. Therefore, there is no uniform pain threshold.

- Heredity, socio-cultural level, energy level, coping skills, and prior experiences with pain define pain tolerance among individuals.

2. Neglect of pain medications at ED when busy in resuscitating the patient.

- Assessment of pain is a necessary, but not a sufficient

component in pain care. Pain score should be monitored with the aim of addressing relief (“correcting” where possible).

- Pain is inevitable whereas suffering is optional, thus emergency care provider’s needs to treat the pain or acknowledge the reason for non-treatment as such should occur both in conversations with the patient (or family) and in the medical record.

3. Failure to anticipate major complications of pain relief medication, e.g. toxicity, anaphylaxis reaction.

- Close monitoring during intravenous administration of pain medication to identify major complication is preferred.
- Early and systematic approach in management of major complication can improve the morbidity and mortality

4. Unrelieved pain has adverse physical and psychological consequences.

- ECP should encourage the reporting of pain by individuals who are reluctant to discuss pain, deny pain when it is likely to present, or fail to follow through on prescribed pain relief medications.

## Special considerations

### Pediatric

Pain management in the pediatric population is challenging and they are at higher risk for under-recognized and under-treated. Neonates and even premature babies can and do feel pain. Pain experienced by children is no less and may even be more than that experienced by an adult. Children react to and report pain in different ways e.g. becomes quiet or withdrawn instead of crying.

The lack of IV access (time-consuming and painful) and unwarranted fears on the

use of pain medication in children especially opioids is a common problem. Therefore, alternative analgesia routes such as nasal medication administration are helpful in younger patients.

### Geriatric

Being an elderly not only had a higher risk for inadequate pain assessment but also to suffer untoward side effects of the pain relief medication especially in the demented patient. ECP need to weight out the risks and benefits of analgesia and should be discussed with patients and family members. It may be relieved through the use of opioid-sparing analgesic regimens or employment of specific therapies (e.g., regional nerve blocks for hip fractures).

### Pregnant patient

Poor acute pain management may lead to chronic pain and is associated with hypertension, anxiety, and depression. Commonly prescribed pain medications are relatively safe in pregnancy. There is no evidence showing analgesics

increases the risk of major malformations. However, NSAIDs should not be used after 32 weeks' gestation because of the possibility of bleeding effects. If opioids are used with caution during the pregnancy, the infant should be observed carefully for any signs of withdrawal. This is called as neonatal abstinence syndrome.

### Drugs seeking behavior patient

Healthcare providers should have a sound understanding of the anatomy, physiology, and psychology of addictive behaviors. A focused history and examination should concentrate on items that can indicate inconsistencies or falsifications associated with inappropriate drug-seeking behavior. It was always difficult as a decision has to be made between “losing” to drug seekers and denying analgesia to patients who are genuinely in need. It is best to give patients the benefit of the doubt with due diligence.



References and Further Reading, click [here](#)

# Paralysing Agents

by Qais Abuagla

## General Information

Paralysis agents are neuromuscular-blocking agents (NMBA). They block neuromuscular transmission at the motor endplate.

## Classification

1. Non-depolarizing blocking agents (NDBA): They act by competitively blocking the binding of acetylcholine to its receptors, e.g., rocuronium
2. Depolarizing blocking agents (DBA): These agents act by depolarizing the motor endplate of the skeletal muscle fiber. This persistent depolarization makes the muscle fiber resistant to further stimulation by acetylcholine, e.g., succinylcholine. It has 2 phases of action – fasciculation and then desensitization.

## Indications

Paralysis drugs in the emergency department are used in rapid sequence intubation (RSI) to produce paralysis, which helps in RSI in 2 ways:

1. paralyze the vocal cords, and permit intubation of the trachea
2. relax the skeletal muscle to facilitate intubation

## Succinylcholine

Succinylcholine is the only DBA used in the ED. It is rapidly hydrolyzed by plasma pseudocholinesterase into weak NMBA. Succinylcholine is rapidly active, typically producing intubating conditions within 45 seconds of administration by rapid intravenous bolus injection.



## Contraindications

- Hyperkalemia
- Preexisting hyperkalemia
- Burns >5 days old
- Crush injury >5 days old
- Severe infection >5 days old
- Neuromuscular diseases (e.g., Myasthenia Gravis)
- History of Malignant Hyperthermia
- Allergy to succinylcholine

## Dosing and Administration

- Adult 1.5 mg/kg IV
- Pediatrics 1.5 mg/kg IV

## Adverse Effects

- Cardiovascular System: Succinylcholine can lead to bradycardia, significant in pediatric patients that are 1 year and less. For that, some practitioners recommend atropine prior to succinylcholine administration, but there is no evidence supporting that.

- Malignant Hyperthermia: is a syndrome characterized by rapid temperature rise and rhabdomyolysis. Treatment for this consists of cessation of any potential offending agents and administration of dantrolene.

## Pregnant Patient Considerations

Category C

## Rocuronium

Rocuronium is one of the NDBA. It works in less than 1 minute after administration.

## Contraindications

- No absolute contraindication to it
- Anaphylaxis

## Dosing and Administration

- Adult 1 mg/kg
- Pediatrics 1 mg/kg

## Adverse Effects

- Hypertension in 1-2%
- Hypotension in 1-2%

## Pregnant Patient Considerations

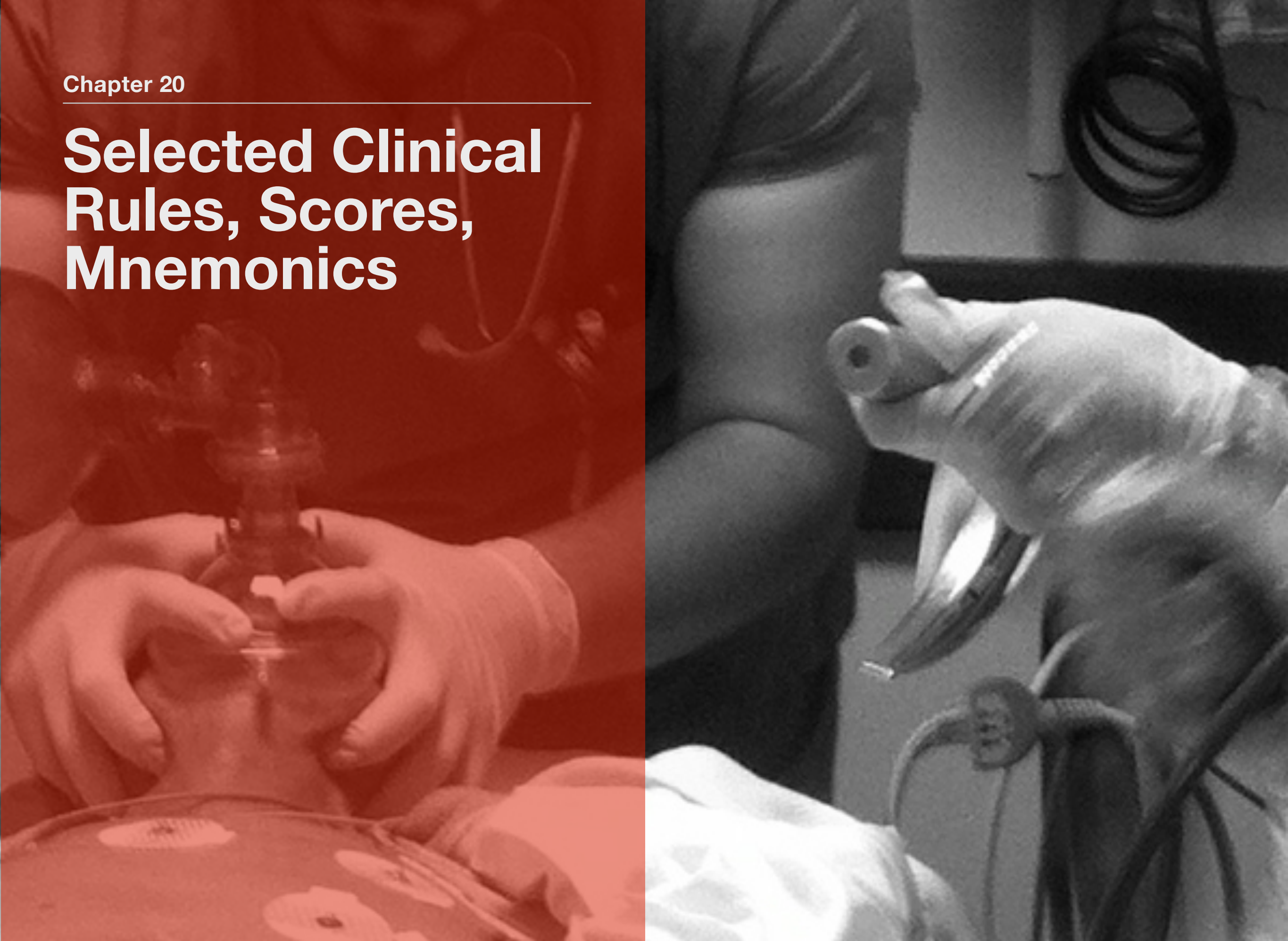
Category C

## Reversal agent

Sugammadex

**References and Further Reading**, click [here](#)

# Selected Clinical Rules, Scores, Mnemonics



# Clinical Decision Rules

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by Stacey Chamberlain

## Introduction

Clinical Decision Rules (CDRs), also known as Decision “Instruments” or “Aids,” are evidence-based tools to assist the practitioner in decision-making for common complaints. In the Emergency Department (ED) setting, these decision aids are often used to help identify patients that might be higher risk for serious conditions such as pulmonary embolism (PE) or subarachnoid hemorrhage (SAH), or they are used to prevent overuse of unnecessary testing, which is how many of the orthopedic rules are applied.

CDRs, despite being called “Rules,” are not meant to replace critical thinking from experienced practitioners. In fact, many of the

CDRs have been directly compared against clinician gestalt or clinical practice, and they do not always fare better. Additionally, some rules actually incorporate clinician gestalt whereas the rule cannot even be applied unless the pre-test probability (based on physician’s judgment of the likelihood of the disease) is below a pre-determined threshold. Also, for a CDR to be useful to a practitioner, it must be practical. If a CDR is developed that has too many complicated variables, it is unlikely to be applied in a busy clinical environment.

Another caveat to the application of CDRs is that they must be applied appropriately. CDRs evolve through a process of derivation to validation to impact analysis of the tool. After the

tool is derived (level 4 evidence), the tool is validated in a limited patient setting (level 3 evidence), then a broader validation setting (level 2 evidence) and finally, the impact of the tool is assessed (level 1 evidence). These levels are important to caution the novice learner against applying every CDR derived and published automatically into their clinical practice. The tool must be validated in a patient population with similar characteristics to the practitioner's patient population. For example, the tool may not perform the same (have the same sensitivity and specificity) if the prevalence of disease is different between the study and actual patient populations. Also, the practitioner must be familiar with the inclusion and exclusion criteria for a particular tool. If not, the tool could be misused. For example, if the tool was derived and validated for a patient population over the age of 18, it should not be inappropriately applied in a pediatric setting.

The practitioner must also understand the purpose of the CDR and whether it is a one-way or two-way rule. As noted by Green, for example, the Ottawa Ankle Rules are intended to be a two-way rule; if the patient meets criteria, you do an X-ray. If they don't meet criteria, you do not do an X-ray. There are two paths you can take after you apply your CDR. Alternatively, the pulmonary embolism rule-out criteria (PERC) demonstrate a one-way rule. This tool was developed to identify a subset of patients at very low risk for PE such that no further testing need be done. If the patient is "PERC positive," this should not imply that further testing for PE such as a D-dimer should be done. Whether or not additional testing should be done remains up to the practitioner and depends on many variables including whether an alternate diagnosis is much more likely. PERC was simply designed to help "rule out" the diagnosis of PE, not "rule in." This rule only guides you down one path, potentially to do no testing; it makes no

judgment as to what you should do if the patient is "PERC positive."

In addition to CDRs, there are many risk stratification tools or scales that are currently used for serious conditions such as pulmonary embolism (PE) and acute coronary syndrome (ACS). Others are being developed for use in the ED setting for common conditions such as congestive heart failure (CHF), chronic obstructive pulmonary disease (COPD) and transient ischemic attack (TIA) to identify patients at higher risk for acute severe complications. From a practical perspective, the ED physician will often use these risk stratification devices to help determine which patients require admission. However, these tools are less prescriptive in that they are not rules that suggest what a practitioner should or should not do; rather, they help the physician more objectively look at the risk for an individual patient. Then the practitioner must decide what level of risk they are comfortable with in regards to inpatient or outpatient management,



which may greatly depend on the resources available in those environments. Most of the risk stratification tools encompass multiple variables with more complicated scoring systems; as they are not easily memorized, most of these would typically be used by ED physicians with real-time access to a computer or smartphone with appropriate apps.

Given the many pitfalls noted above regarding CDRs, the goals of using evidence-based medicine to reduce practice variability, maximize use of resources, and help identify and diagnose high-risk conditions are important. It is equally important that the ED physician critically appraise these tools and selectively apply them in appropriate ways. The remainder of this chapter will use case scenarios to review the most commonly used CDRs in the ED setting.

The useful FOAM reference [MDCalc.com](http://MDCalc.com) provides a summary of the most common tools that are being used with easy-to-

use online calculators, additional information on inclusion and exclusion criteria, and pearls and pitfalls for each tool.

## Orthopedic CDRs

### Case 1

*A 28-year-old man presents to the ED with left ankle pain after twisting his ankle playing basketball. He is able to bear weight and notes pain and swelling to the lateral aspect of the ankle (he points to just below the lateral malleolus). He denies weakness, numbness, or tingling and has no other injuries. On exam, he is neurovascularly intact. Edema and tenderness are noted slightly anterior and inferior to the lateral malleolus. There is*

*no point tenderness to the distal posterior malleoli bilaterally.*

**Should you get an X-ray to rule out fracture?**

### Ottawa Ankle Rule

- Pain in the malleolar zone and any one of the following:
- Bone tenderness along the distal 6 cm of the posterior edge or tip of the tibia (medial malleolus), OR
- Bone tenderness along the distal 6 cm of the posterior edge or tip of the fibula (lateral malleolus), OR
- An inability to bear weight both immediately after the trauma and in the ED for four steps.

### Ottawa Foot Rule

- Pain in the midfoot zone and any one of the following:

- Bone tenderness at the base of the fifth metatarsal, OR
- Bone tenderness at the navicular bone, OR
- An inability to bear weight both immediately after the trauma and in the ED for four steps.

### Ottawa Knee Rule

- Knee injury with any of the following:
  - Age 55 years or older
  - Tenderness at head of fibula
  - Isolated tenderness of patella
  - Inability to flex to 90°
  - Inability to bear weight both immediately after the trauma and in the ED (4 steps)

### Pittsburgh Knee Decision Rule

- Mechanism: blunt trauma or fall
- Age < 12 or > 50
- Unable to bear weight 4 steps in the ED

Some of the longest standing and most widely accepted CDRs are the Ottawa knee, ankle, and foot rules. These rules are to help practitioners identify patients with an extremely low risk of fracture such that X-rays do not need to be done, thus limiting the risks and costs of unnecessary testing. The sensitivity of these rules has been found to be 98.5-100%. In impact study of the Ottawa knee rule, application of the rule decreased the use of knee radiography without patient dissatisfaction or missed fractures and was associated with reduced waiting times and costs. These rules have been validated in pediatric populations as well with similar sensitivities (98.5-100%).

A less studied rule for knee trauma to determine the need for radiography is the Pittsburgh Decision Rule (PDR). It differs from the Ottawa rule in that it looks at the mechanism of injury and applies to a different age group; also, point tenderness is not used in the PDR. Its original derivation study found this rule to

be 100% sensitive. Two studies compared the PDR and Ottawa knee rules and found the PDR to perform better with similar sensitivities but better specificity for the PDR (51-60% versus 27%). However, one validation study for the PDR found the sensitivity to be as low as 77%. Additionally, while the Ottawa rule has been validated in children as young as two years old, the PDR excludes children younger than 12.

### Case 1 Discussion

In the above case, using either CDR, an X-ray is unnecessary.

### Trauma CDRs

#### Case 2

*A 57-year-old man fell from a height of 12 feet while on a ladder. He did not pass out; he reports that he simply lost his footing. He fell onto a grassy area, hitting his head and*

*complains of neck pain. He did not lose consciousness and denied headache, blurry vision, vomiting, weakness, numbness or tingling in any extremities. He denies other injuries. He was able to get up and ambulate after the fall and came in by private vehicle. He has not had previous spine surgery and does not have known vertebral disease. On exam, he is neurologically intact with a GCS of 15, does not appear intoxicated and has moderate midline cervical spine tenderness.*

**Should you get imaging to rule out a cervical spine fracture?**

## Canadian C-spine Rule

- Age  $\geq$  65
- Extremity paresthesias
- Dangerous mechanism (fall from  $\geq$  3ft / 5 stairs, axial load injury, high-speed MVC/rollover/ejection, bicycle collision, motorized recreational vehicle)

## NEXUS Criteria for C-spine Imaging

- Focal neurologic deficit present
- Midline spinal tenderness present
- Altered level of consciousness present
- Intoxication present
- Distracting injury present

Both the Canadian C-spine Rule (CCR) and NEXUS Criteria are widely employed in clinical practice to reduce unnecessary cervical spine imaging in trauma patients with neck pain or obtunded trauma patients. The CCR uses mechanism and age criteria, whereas the NEXUS Criteria incorporates criteria including midline

tenderness and additional factors that might limit a practitioner's exam. The CCR can be difficult for some practitioners to remember all the criteria that qualify as a dangerous mechanism and is limited to ages  $> 16$  and  $< 65$ . However, it can be used in intoxicated patients if the patients are alert and cooperative, allowing a full neurologic exam. The NEXUS Criteria are applicable over any age range ( $> 1$  year old), but the sensitivity may be low in patients  $> 65$  years of age. A single comparison study found the CCR to have better sensitivity (99.4% versus 90.7%); however, the study was performed by hospitals involved in the initial CCR validation study.

## Case 2 Discussion

By applying either criteria to this case, the patient would require C-spine imaging as by CCR, the patient would meet criteria for dangerous mechanism, and by NEXUS, the patient has midline tenderness to palpation.

### Case 3

*A 36-year-old woman slipped on ice and fell and hit her head. She reports loss of consciousness for a minute after the event, witnessed by a bystander. She denies headache. She denies weakness, numbness or tingling in her extremities and no changes in vision or speech. She has not vomited. She remembers the event except for the transient loss of consciousness. She doesn't use any blood thinners. On physical exam, she has a GCS of 15, no palpable skull fracture and no signs of a basilar skull fracture.*

**Should you get a CT head for this patient to rule out a clinically significant brain injury?**

### Canadian CT Head Rule

- High-Risk Criteria (rules out the need for neurosurgical intervention)
  - GCS < 15 at two hours post-injury
  - Suspected open or depressed skull fracture
  - Any sign of basilar skull fracture (hemotympanum, Raccoon eyes, Battle's sign, CSF oto or rhinorrhea)
- Medium Risk Criteria (rules out clinically important brain injury)
  - Retrograde amnesia to event  $\geq$  30 minutes
  - Dangerous mechanism (pedestrian struck by motor vehicle, ejection from the motor vehicle, fall from > 3 feet or > 5 stairs)

The Canadian CT Head Rule (CCHR) only applies to patients with an initial GCS of 13-15, witnessed loss of consciousness (LOC), amnesia to the head injury event, or confusion. The study was only for patients > 16 years of age. Patients were excluded from the study if they had "minor head injuries" that didn't even meet these criteria. Patients were also excluded if they had signs or symptoms of moderate or severe head injury including GCS < 13, post-traumatic seizure, focal neurologic deficits, or coagulopathy. Other studies have looked at different CDRs for traumatic brain injury including the New Orleans Criteria (NOC). However, CCHR has been found to have superior sensitivity and specificity.

### Case 3 Discussion

By applying this rule to the above case, the patient should be considered for imaging due to the mechanism. A fall from standing for an adult patient would constitute a fall from > 3 feet; therefore,



although the patient would not likely be high risk and need neurosurgical intervention, the patient might have a positive finding on CT that in many practice settings would warrant an observation admission.

#### Case 4

*A 20-month-old female was going up some wooden stairs, slipped, fell down four stairs, and hit the back of her head on the wooden landing at the bottom of the stairs. She did not lose consciousness and cried immediately. She was consolable after a couple of minutes and is acting normal per her parents. She has not vomited. On exam, she is well-appearing, alert, and has a normal neurologic exam. She is noted to have a left parietal*

*hematoma measuring approximately 4×4 cm.*

**Should you get CT imaging of this child to rule out clinically significant head injury?**

#### PECARN Pediatric Head Trauma Algorithm

- Age < 2
  - GCS < 15, palpable skull fracture, or signs of altered mental status
  - Occipital, parietal or temporal scalp hematoma; History of LOC ≥ 5 sec; Not acting normally per parent or Severe Mechanism of Injury?
- Age ≥ 2
  - GCS < 15, palpable skull fracture, or signs of altered mental status
  - History of LOC or history of vomiting or Severe headache or Severe Mechanism of Injury?

The PECARN (Pediatric Emergency Care Applied Research Network) Pediatric Head Trauma Algorithm was developed as a CDR to minimize unnecessary radiation exposure to young children. The estimated risk of lethal malignancy from a single head CT in a 1-year-old is 1 in 1000-1500 and decreases to 1 in 5000 in a 10-year-old. Due to these risks, in addition to costs, length of stay and potential risks of procedural sedation, this CDR is widely employed given the frequency of pediatric head trauma ED visits. This CDR has the practitioner use a prediction tree to determine risk, but unlike some other risk stratification tools, the PECARN group does make recommendations based on what they consider acceptable levels of risk. In the less than 2-year-old group, the rule was found to be 100% sensitive with sensitivities ranging from 96.8%-100% sensitive in the greater than two-year-old group.

This algorithm does have some complexity and ambiguity. It requires the

practitioner to know what were considered signs of altered mental status and what were considered severe mechanisms of injury. In addition, certain paths of the decision tree lead to intermediate risk zones. In these cases, the recommendation is “observation versus CT,” allowing for the ED physician to base his/her decision to image or not based on numerous contributory factors including physician experience, multiple versus isolated findings, and parental preference, among others.

Other pediatric head trauma CDRs rules have been derived and validated; however, in comparison trials, PECARN performed better than the other CDRs.<sup>1</sup> Of note, in this study, physician practice (without the use of a specific CDR) performed as well as PECARN with only slightly lower specificity.

## Case 4 Discussion

For purposes of the case study, the patient falls into an intermediate risk zone of clinically important brain injury.

However, a sub-analysis of patients less than two years old with isolated scalp hematomas suggests that patients were higher risk if they were < 3 months of age, had non-frontal scalp hematomas, large scalp hematomas (> 3cm), and severe mechanism of injury. Given the large hematoma in the case study patient and a severe mechanism of injury (a fall of > 3 feet in the under two age group), one might more strongly consider imaging due to these two additional higher risk factors.

## PECARN Abdominal Trauma

- Evidence of abdominal wall trauma/seatbelt sign or GCS < 14 with blunt abdominal trauma (if no, go to next point)
  - 5.4% risk of needing intra-abdominal injury intervention
- Abdominal tenderness (if no, go to next point)
  - 1.4 % risk of intra-abdominal injury intervention

- Thoracic wall trauma, complaints of abdominal pain, decreased breath sounds, vomiting

- 0.7% risk of intra-abdominal injury intervention

A CDR for pediatric blunt abdominal trauma has been derived by the PECARN group but not yet validated. This CDR uses a seven-point decision rule. If the patient does not have any of these findings, the patient would be considered “very low risk” with a 0.1% risk of intra-abdominal injury intervention required. A study did compare the PECARN CDR versus clinical suspicion and found that the CDR had significantly higher sensitivity (97.0% vs. 82.8%) but lower specificity (42.5% vs. 78.7%). However, abdominal CTs were done in 33% of patients with clinical suspicion < 1%, meaning that even though clinical suspicion had higher specificity, this often did not translate into clinical practice. Validation of the PECARN rule has the potential to therefore improve both

sensitivity and specificity compared to physician practice, but this remains to be seen.

## Additional CDRs

### Case 5

*A 24-year-old woman presents with headache that began three hours prior to arrival to the ED. The patient was at rest when the headache began. The headache was not described as “thunderclap,” but it did reach maximum severity within the first 30 minutes. The headache is generalized and rated 10/10. She denies head trauma, weakness, numbness, and tingling in her extremities. She denies visual changes, changes in speech and neck pain. She has not taken*

*anything for the headache. She does not have a family history of cerebral aneurysms or polycystic kidney disease. On physical exam, she has a normal neurologic exam and normal neck flexion.*

**Should you do a head CT and/or a lumbar puncture to evaluate for a subarachnoid hemorrhage in this patient?**

### Ottawa SAH Rule

- Investigate if  $\geq 1$  high-risk variables present:
- Age  $\geq 40$
- Neck pain or stiffness
- Witnessed loss of consciousness
- Onset during exertion
- Thunderclap headache (instantly peaking pain)
- Limited neck flexion on exam

A CDR to determine risk for subarachnoid hemorrhage (SAH) was derived and has been externally validated in a single study. The CDR’s purpose was to identify those at high risk for SAH and included those with acute non-traumatic headaches that reached maximal intensity within one hour and who had normal neurologic exams. Of note, the rule has many inclusion and exclusion criteria that the ED physician must be familiar with and was only derived for patients 16 years or older. The study authors note that the CDR is to identify patients with SAH; it is not an acute headache rule. In the validation study, of over 5,000 ED visits with acute headache, only 9% of those met inclusion criteria. Also, clinical gestalt again plays a role as the authors suggest not to apply the CDR to those who are ultra-high risk with a pre-test probability for SAH of  $> 50\%$ .

The Ottawa SAH Rule was 100% sensitive but did not lead to reduction of testing vs. current practice. The authors state that the value of the Ottawa SAH

Rule would be to standardize physician practice in order to avoid the relatively high rate of missed sub-arachnoid hemorrhages.

### Case 5 Discussion

By applying the Ottawa SAH Rule, this patient is low risk and does not require further investigation for a SAH.

### Case 6

*A 19-year-old female presents with sharp right flank pain and shortness of breath that started suddenly the day prior to arrival. The pain is worse with deep inspiration but not related to exertion and not relieved with ibuprofen. She denies anterior chest pain, cough, and fever. She denies leg pain or swelling and recent travel, immobilization, trauma, or*

*surgery. She has no anterior abdominal pain, no dysuria or hematuria and no personal or family history of gallstones, kidney stones, or blood clots. She's never had this pain before, has no significant past medical history and her only medication is birth control pills. On exam, her vital signs are within normal range, she has normal cardiac and pulmonary exams, no costovertebral angle tenderness, no chest wall or abdominal tenderness and no leg swelling.*

**Do you need to do any studies to evaluate this patient for a pulmonary embolism?**

### Pulmonary Embolism Rule-Out Criteria (PERC)

- Age  $\geq 50$
- Heart rate  $\geq 100$
- O<sub>2</sub> sat on room air  $< 95\%$
- Prior history of venous thromboembolism
- Trauma or surgery within 4 weeks
- Hemoptysis
- Exogenous estrogen
- Unilateral leg swelling

The PERC CDR was originally derived and validated in 2004 and with a subsequent multi-study center validation in 2008. In the larger validation study, the rule was only to be applied in those patients with a pre-test probability of  $< 15\%$ , therefore incorporating clinical gestalt prior to using the rule. PERC is a one-way rule, as mentioned above, which tried to identify patients who are so low-risk for pulmonary embolism (PE) as to



not require any testing. It does not imply that testing should be done for patients who do not meet criteria, and it is not meant for risk stratification, as opposed to the Wells' and Geneva scores.

### Case 6 Discussion

In order to apply the PERC CDR to the case study patient, the ED physician pre-supposes a pre-test probability of < 15%. If the ED physician has a higher pre-test probability than that, he/she should not use the PERC CDR. If the ED physician, in this case, did indeed have a pre-test probability of < 15%, the case study patient would fail the rule-out due to her use of oral contraceptives. In that case, the ED physician would need to determine if he/she would do further testing which could include a D-dimer, CT chest with contrast, ventilation/perfusion scan, or lower extremity Doppler studies to evaluate for deep vein thromboses (DVTs). The PERC CDR gives no guidance in this case.

## Risk Stratification Tools

### Case 7

*A 68-year-old male presents with acute onset of shortness of breath and right-sided sharp chest pain, worse with deep breathing. He denies chest pain with exertion, no cough, fever or hemoptysis, no leg pain or swelling, no recent travel, surgery or immobilization. He has a history of prostate cancer and completed his treatment with radiation therapy four months ago. On exam, his heart rate is 90, O2 saturation is 98% with a normal respiratory rate, blood pressure, and temperature. His cardiac and pulmonary exams*

*are normal, and there is no calf tenderness or swelling.*

**How should you proceed with this patient's work up for PE?**

**Table 20.1** Wells' Criteria for Pulmonary Embolism

CRITERIA	POINT VALUE
Clinical signs and symptoms of DVT	+3
PE is #1 diagnosis, or equally likely	+3
Heart rate > 100	+1.5
Immobilization at least 3 days, or Surgery in the Previous 4 weeks	+1.5
Previous, objectively diagnosed PE or DVT	+1.5
Hemoptysis	+1
Malignancy w/ Treatment within 6 mo, or palliative	+1

**Table 20.2** Geneva Score (Revised) for Pulmonary Embolism

CATEGORY	CRITERIA	POINT VALUE
Risk factors	Age > 65	+1
	Previous DVT or PE	+3
	Surgery (under general anesthesia) or lower limb fracture in past 1 month	+2
	Active malignant condition	+2
Symptoms	Unilateral lower limb pain	+3
	Hemoptysis	+2
Signs	Heart rate < 75	0
	Heart rate 75 - 94	+3
	Heart rate ≥ 95	+5
	Pain on lower limb deep venous palpation and unilateral edema	+4

The Wells’ Criteria for PE is a risk stratification score with different point values assigned to different criterion. Its purpose is to identify patients who have a lower risk for PE in order to potentially avoid unnecessary testing and the risks and costs associated with it. The criteria have been validated in the ED setting. In the initial three-tier model, a patient with 0-1 points was considered to be in a low-risk group (1.3% prevalence of PE in an ED population) versus patients with a moderate score of 1-6 ( 16.2% prevalence), and those with a high score of >6 ( 37.5% prevalence). Subsequent studies have been done to apply a simplified version of the Wells’ Criteria and also to use the Wells’ Criteria along with D-dimer testing in a dichotomous manner (two-tier model) where a score of 4 or less (“PE Unlikely” group) combined with a negative D-dimer would achieve sufficiently low probability of PE so as not to pursue further workup. This two-tier model is supported by the American College of Physicians (ACEP) Clinical Guidelines. A two-tier model using a cut-

off of less than 6 for low risk was studied in pregnant patients with a negative predictive value of 100%.

The original Geneva score included the use of chest radiography and an ABG, whereas the revised score (rGeneva) uses only clinical criteria. A patient with a rGeneva score of 0-3 is considered the low risk with a < 10% prevalence of PE. A score of 4-10 identifies intermediate-risk patients, and a score of 11+ is high risk (>60% prevalence or PE).

The Wells and rGeneva scores have been compared and found to have overall similar accuracy. These PE risk stratification tools are meant to be applied in those patients with concern for PE as a diagnosis. If PE is not under consideration, the tools should not be applied. Practically speaking, for many ED physicians, these tools are used to help risk stratify patients to identify those who are very low-risk such that no testing should be done, low to intermediate risk such that D-dimer testing would be a

useful diagnostic tool, or high risk such that even if a D-dimer were negative, the post-test probability would remain high enough that further testing should be pursued. One recent study found that physician gestalt actually performed better than either the Wells or rGeneva scores. However, guidelines from the Clinical Practice Committee of the American College of Physicians (ACP) were published in 2015 that outline best practice advice including advocating that clinicians should use validated CPRs to estimate pre-test probability in patients in whom acute PE is being considered.

### Case 7 Discussion

This patient's Wells' score is 4. Although subject to ED physician judgment, PE could be considered at least equally as likely as any other diagnosis given the absence of other findings to explain his shortness of breath (no crackles or wheezing on exam, no cough or fever). The patient's rGeneva score is 6. An "active malignant condition" is defined as

a "solid or hematologic malignant condition, currently active or considered cured < 1 year." Using the dichotomous Wells' approach, the patient would be considered "PE Unlikely;" using the rGeneva, the patient would be intermediate risk. The ACP Guidelines would suggest that a D-dimer should be done in this patient, adjusted for age, to determine the need for possible imaging to evaluate for PE.

### Case 8

*A 50-year-old male presents to the ED complaining of chest pain for two days. His pain is substernal, non-radiating, described as a tightness, not related to exertion. He has no associated shortness of breath, nausea or diaphoresis. No cough or fever. He's never had this pain before. He has a history of hypertension but no*

*other cardiac risk factors.*

*His exam in the ED is normal, and his EKG and initial troponin are normal.*

**Does this patient require additional cardiac workup in the ED or admission to hospital for additional workup? Can this patient be safely discharged for outpatient follow-up?**

The HEART Score is used to risk stratify chest pain patients in the ED to identify those at risk for major adverse cardiac events (MACE) within six weeks. With the HEART Score, low-risk patients have a score of 0-3 and have a less than 2% risk of MACE at 6 weeks. The HEART Score differs from the Thrombolysis in Myocardial Infarction (TIMI), and Global Registry of Acute Coronary Events (GRACE) scores as those scores measure the risk of death for patients with diagnosed acute coronary syndromes (ACS) rather than identifying patients who have cardiac-related chest pain in the first

place. Additionally, even with low TIMI scores for those diagnosed with ACS in the ED, there is still a 4.7% risk of a bad outcome. This may be of little utility to the

ED physician who finds this risk level unacceptable.

**Table 20.3** HEART Score for Cardiac Events

CATEGORY	CRITERIA	POINT VALUE
History	Highly suspicious	+2
	Moderately suspicious	+1
	Slightly suspicious	0
EKG	Significant ST depression	+2
	Non specific repolarization disturbance	+1
	Normal	0
Age	≥ 65	+2
	45-65	+1
	≤ 45	0
Risk Factors (include: hypercholesterolemia, hypertension, diabetes mellitus, cigarette smoking, positive family history, obesity)	≥ 3 risk factors or history of atherosclerotic disease	+2
	1-2 risk factors	+1
	No risk factors known	0
Troponin	≥ 3× normal limit	+2
	1-3× normal limit	+1
	≤ normal limit	0

## Case 8 Discussion

This patient's HEART Score is 3 if the physician considers the history "moderately suspicious." The patient is at low risk for a major cardiac event in the next six weeks so that the ED physician could consider outpatient follow-up. Again, however, the risk stratification scores are not prescriptive, however. Decision-making must be done by the clinician based on his/her judgment, resources available, and comfort with certain levels of risk.

## Case 9

*A four-year-old boy presents to the ED with a complaint of sore throat for one day associated with cough and fever. On exam, he is febrile to 38.5 degrees Celsius, has bilateral tonsillar exudates, and anterior cervical lymphadenopathy.*



## How should you proceed with the workup for this child for possible strep?

*Options include treating empirically, doing a rapid point-of-care strep test, sending a throat culture, or supportive treatment.*

The Centor Score is a risk stratification tool to look at clinical criteria that suggest a greater likelihood of strep pharyngitis that may prompt the ED physician to prescribe antibiotics. It was originally designed for use in adults, but a modified score has been validated for use in children > 2 years of age and adults that includes age criteria as strep pharyngitis is a more common condition in children. In the absence of any of the criteria at any age group, the risk of strep is less than 10% (< 2.5% if 15 or older) and further testing is not necessary. With a score of 4 or more points, the probability

**Table 20.4** Centor Score (Modified) for Streptococcal Pharyngitis

CATEGORY	CRITERIA	POINT VALUE
Age	3-14	+1
	15-44	0
	45 or older	-1
Exudate or swelling on tonsils		+1
Tender/swollen anterior cervical lymph nodes		+1
Fever (T > 38°C, 100.4°F)		+1
Cough	Present	0
	Absent	+1

of strep is greater than 50%, and some would advocate for empiric antibiotics in this group. However, as a risk stratification tool, ED physicians can adjust their practice according to their interpretation of the risks.

Antibiotics have been shown to reduce suppurative (peritonsillar abscess, cervical lymphadenitis, and mastoiditis) and non-suppurative (e.g., acute rheumatic fever) complications of strep pharyngitis and shorten the duration of clinical symptoms as well as reducing transmission. Rapid antigen detection tests have been found to have a sensitivity between 70 and 90% and a specificity of  $\geq 95\%$ . Some authors recommend rapid antigen detection testing (RADT) only for children with high clinical scores (using Centor or other published clinical criteria) or if the results of the standard throat culture will not be available for more than 48 hours. Additionally, the presence of particular clinical criteria may impact the ED physician's decision to test and/or treat. Studies looking at different clinical prediction scores (including Centor) found that the presence of tonsillar exudates conferred the highest odds of having streptococcus infection.

## Case 9 Discussion

The patient has a Centor Score of 4. Some clinicians would use this high-risk clinical score to justify further testing with an RADT or a throat culture. Others would treat empirically, especially given the presence of exudates which has a higher specificity than some of the other clinical findings. This decision may be based on additional factors such availability and processing times of diagnostic testing and ease of patient follow-up.

### Case 10

*A 30-year-old male presents to the ED with nausea, vomiting, and epigastric discomfort for one day. He vomited multiple times, initially non-bloody, then developed some blood in the vomit during the last two episodes, which he quantified as a teaspoon in each. He denies melena or hematochezia. He has no*

*diarrhea, fever, or syncope. He denies a history of liver or heart problems. On exam, he has normal vital signs with an initial blood pressure of 128/78 in the ED, and his abdomen is non-tender. His hemoglobin is 13.5, and BUN is 5.*

**Does this patient need admission for further monitoring or evaluation of his upper GI bleed?**

Glasgow-Blatchford Risk Score is useful for predictive of inpatient mortality, blood transfusions, re-bleeding, ICU monitoring, and hospital length of stay. Patients with a score of zero may be discharged home, those with score 2 or higher are usually admitted, and those with score of 10 or more are at highest risk for morbidity and resource utilization. Maximum score is 23.

**Table 20.5** Glasgow-Blatchford Risk Score

CATEGORY	SCORE
BUN in mg/dL	
18.2 to 22.4	2
22.5 to 28	3
28.1 to 70	4
70.1 or greater	6
Hemoglobin, men g/dL	
12 to 13	1
10 to 11.9	3
9.9 or less	6
Hemoglobin, women g/dL	
10 to 12	1
9.9 or less	6
Systolic Blood Pressure, mmHg	
100-109	1
90-99	2
<90	3
Heartrate >100 peats per minute	1
Melena	1
Syncope	2
Hepatic Diseases	2
Heart failure	2

Glasgow-Blatchford Risk Score is useful for predictive of inpatient mortality, blood transfusions, re-bleeding, ICU monitoring, and hospital length of stay. Patients with a score of zero may be discharged home, those with score 2 or higher are usually admitted, and those with score of 10 or more are at highest risk for morbidity and resource utilization. Maximum score is 23.

The Glasgow-Blatchford Bleeding Score (GBS) uses clinical information as well as some diagnostic testing to risk stratify upper GI bleeding patients. It should not be used for lower GI bleeding patients or patients in whom the source of GI bleeding is unclear. A score of 0 is considered low risk. Any score higher than 0 is high risk for needing a medical intervention of transfusion, endoscopy, or surgery; therefore, the presence of any of the above criteria would be considered high risk. The tool assigns different point values to different gradations of the variables present to a possible highest possible score of 29.

## Case 10 Discussion

The patient does not meet any of the criteria in the GBS and would be considered low risk. The patient does not demonstrate any signs of lower GI bleeding and could likely be safely discharged home based on this risk stratification.

## Case 11

*A 45-year-old woman presents with syncope immediately prior to arrival. She was feeling generalized fatigue prior to the syncopal episode. She denies chest pain, palpitations, or shortness of breath. She has not had vomiting or diarrhea. She has been taking PO today but has a decreased appetite. She has no known medical problems. On exam, her initial*

*BP is 86/48 which improves to 98/50 with 1L IVF. Her hematocrit is 31%, and her EKG and telemetry monitoring in the ED are normal.*

**Is this patient low risk for safe discharge home?**

## San Francisco Syncope Rule

- Congestive heart failure history
- Hematocrit < 30%
- EKG abnormal (new EKG change from any source, any non-sinus rhythm on EKG or monitoring)
- Shortness of breath symptoms
- Systolic BP < 90 mmHg at triage

The San Francisco Syncope Rule was derived in 2004. In its initial derivation and validation studies, it was found to have 92% and 98% sensitivity, respectively. Its use has become

controversial, however, due to inconsistent validation studies where it has not performed as well. A systematic review of the literature from 2011 suggested that “the probability of a serious outcome given a negative score with the San Francisco Syncope Rule was 5% or lower, and the probability was 2% or lower when the rule was applied only to patients for whom no cause of syncope was identified after initial evaluation in the emergency department.” However, a meta-analysis from 2013 suggests that it only had 87% sensitivity for serious outcomes according to pooled results and that there was a broad range of false-negative rates among the included studies (range 0% to 48%). Although there is clearly no consensus on use of this tool to safely discharge patients with syncope home, if they do not meet these criteria, patients who do have criteria would be considered higher risk, possibly warranting observation, admission and/or further diagnostic studies.

### Case 11 Discussion

Although the San Francisco Syncope Rule has failed to be consistently validated for use in identifying all high-risk patients, this patient fails the rule due to her initial SBP being less than 90. Therefore, this patient would, in any case, not be considered low-risk, and the ED physician might consider additional monitoring and/or evaluation.

### Ottawa Heart Failure Risk Score, COPD Risk Scale and Canadian TIA Risk Score

These risk stratification tools are mentioned as they have all completed derivation studies and are in various stages of validation studies. The intent of these tools is to help the clinician develop risk estimates of short-term serious adverse events in ED patients. Although not yet ready for widespread usage, the ED physician should be aware of these. Additional studies need to be completed and published to determine the validity and impact of these scores, but they

certainly have the potential to be useful adjuncts for the management of ED patients with these common conditions.

**References and Further Reading,** click [here](#)



# Mnemonics

by Ozlem Dikme

## 3 D's: Beck's triad (cardiac tamponade)

D = Distant heart sounds

D = Distended jugular veins

D = Decreased arterial pressure

## 4 P's: Arterial occlusion

P = Pain

P = Pallor

P = Pulselessness

P = Paresthesias

## ABC/2: Volume of intracranial bleed of CT

A = Maximal diameter of the hematoma by CT

B = Diameter 90° to A, and

C = Approximate number of CT slices with hemorrhage multiplied by the slice thickness

Vol > 20 – 30 ml consider surgery

## ABCDE: Supraventricular tachycardia (treatment)

A = Adenosine

B = Beta-blocker

C = Calcium channel antagonist

D = Digoxin

E = Excitation (vagal stimulation)

## ABCDEFGHIJK: Haematuria differential in children

A = Anatomy (cysts, etc)

B = Bladder (cystitis)

C = Cancer (Wilm's tumour)

D = Drug related (cyclophosphamide)

E = Exercise induced

F = Factitious (Munchausen by proxy)

G = Glomerulonephritis

H = Haematology (bleeding disorder, sickle cell)

I = Infection (UTI)

J = inJury (trauma)

K = Kidney stones (hypercalciuria)

## ABC HELP: Causes of ST Elevation in ECG

A = AMI

B = Brugada

C = CNS Pathologies

H = Hypertrophy (LVH)

E = benign Early repolarization

L = LBBB

P = Pericarditis

## AEIOU TIPS: Causes of altered mental status

A = Alcohol

E = Epilepsy, electrolytes

I = Infection

O = Overdose

U = Urea

T = Trauma

I = Insulin

P = Psychiatric

S = Sepsis, shock

## APGAR: System to evaluate newborn's condition

A = Appearance (color)

P = Pulse (heart rate)

G = Grimace (reflex, irritability)

A = Activity (muscle tone)

R = Respiratory effort

## APPENDICITIS: RLQ pain differential

A = Appendicitis/ Abscess

P = PID/ Period

P = Pancreatitis

E = Ectopic/ Endometriosis

N = Neoplasia

D = Diverticulitis

I = Intussusception

C = Crohns Disease/ Cyst (ovarian)

I = IBD

T = Torsion (ovary)

I = Irritable Bowel

S = Syndrome Stones

## ASCLAST: Eliciting history of present illness and exploring symptoms

A = Aggravating and alleviating factors

S = Severity

C = Character, quality

L = Location

A = Associated

S = Setting

T = Timing

## ASTHMA: Common Medications used to treat Asthma

A = Albuterol

S = Steroid

T = Theophylline

H = Humidified Oxygen

M = Magnesium (MgSO<sub>4</sub>)

A = Antileukotrienes

## BATS: Subarachnoid hemorrhage causes

B = Berry aneurysm

A = Arteriovenous malformation / Adult polycystic kidney disease

T = Trauma (e.g., being struck with baseball bat)

S = Stroke

## BE FEVEER: Duke's Criteria for Bacterial Endocarditis (BE)

### Major Criteria

B = persistent Blood culture positive >2 times 12 hr part (each C&S taking should be one hour apart, and 3 samples should be taken)

E = Endocardial involvement from Echo

### Minor criteria

F = Fever  $\geq 38^{\circ}\text{C}$

E = Echo findings not fulfilling a major

V = Vascular (vasculitis) – Janeway lesions, mycotic aneurysm, etc

EE = Evidences from microbiological/immunology (2)

R = Risk factors/predisposing factors – drug abuse, valvular diseases (predisposing factors)

## BOOMAR: MI basic management

B = Bed rest

O = Oxygen

O = Opiate

M = Monitor

A = Anticoagulate

R = Reduce clot size

## CARDIAC RIND: Pericarditis causes

C = Collagen vascular disease

A = Aortic aneurysm

R = Radiation

D = Drugs (such as hydralazine)

I = Infections

A = Acute renal failure

C = Cardiac infarction

R = Rheumatic fever

I = Injury

N = Neoplasms

D = Dressler's syndrome

## CAT MUD PILES: Causes of High AG Metabolic Acidosis

C = Carbon monoxide, Cyanide

A = Alcoholic ketoacidosis

T = Toluene

M = Methanol

U = Uremia

D = Diabetic ketoacidosis

P = Paraldehyde, Phenformin

I = Iron, Isoniazid

L = Lactic acidosis

E = Ethylene glycol

S = Salicylates poisoning

## CHADS2: Risk factors for developing stroke in patients with nonrheumatic atrial fibrillation

C = Congestive Heart Failure

H = Hypertension or treated hypertension

A = Age 75 years or older

D = Diabetes Mellitus

S = Stroke or TIA previously

## CHESS: San Francisco Syncope Rule

C = history of Congestive heart failure,

H = Hematocrit  $<30\%$ ,

E = Electrocardiogram abnormality,

S = Shortness of breath, or

S = Systolic blood pressure  $<90$  mm Hg

## CLADE SPADE: Fall potential causes

C = Cardiovascular/ Cerebrovascular

L = Locomotor (skeletal, muscular, neurological)

A = Ageing (increased body sway, decreased reaction time)  
 D = Drugs (esp. antihypertensives, antipsychotics)  
 E = Environmental  
 S = Sensory deficits (eg. visual problems)  
 P = Psychological/ Psychiatric (depression)  
 A = Acute illness  
 D = Dementia  
 E = Epilepsy

## DCAP – BTLS: Things to look for in head-to-toe survey for trauma

D = Deformity  
 C = Contusions  
 A = Abrasions  
 P = Punctures/penetrations  
 B = Burns  
 T = Tenderness  
 L = Lacerations  
 S = Swelling

## DKA precipitants (5 I's)

Infection  
 Ischaemia (cardiac, mesenteric)

Infarction  
 Ignorance (poor control)  
 Intoxication (alcohol)

## DEMENTIA: Dementia, some common causes

D = Diabetes  
 E = Ethanol  
 M = Medication  
 E = Environmental (e.g., CO poisoning)  
 N = Nutritional  
 T = Trauma  
 I = Infection  
 A = Alzheimer's

## DEPRESSED ST: Depressed ST-segment (causes)

D = Drooping valve (MVP)  
 E = Enlargement of LV with strain  
 P = Potassium loss (hypokalemia)  
 R = Reciprocal ST- depression (in I/W AMI)  
 E = Embolism in lungs (pulmonary embolism)  
 S = Subendocardial ischemia  
 S = Subendocardial infarct  
 E = Encephalon haemorrhage (intracranial

haemorrhage)

D = Dilated cardiomyopathy

S = Shock

T = Toxicity of digitalis, quinidine

## DOPE: Acute Deterioration in Intubated Patient

D = Displacement of the tube

O = Obstruction of the tube

P = Patient (this is the first priority, not the machines), Pneumothorax

E = Equipment failure

## DOTS: Signs to suspect fracture

D = Deformity

O = Open wound

T = Tenderness

S = Swelling

## ELEVATION: ST elevation causes in ECG

E = Electrolytes

L = LBBB

E = Early repolarization

V = Ventricular hypertrophy

A = Aneurysm



T = Treatment (e.g., pericardiocentesis)  
 I = Injury (AMI, contusion)  
 O = Osborne waves (hypothermia)  
 N = Non-occlusive vasospasm

## **FAILURE: CHF causes of exacerbation**

F = Forgot medication  
 A = Arrhythmia/ Anaemia  
 I = Ischemia/ Infarction/ Infection  
 L = Lifestyle: taken too much salt  
 U = Upregulation of CO: pregnancy, hyperthyroidism  
 R = Renal failure  
 E = Embolism: pulmonary

## **FAST HUG: Interventions for critically ill patients in ED**

F = Fluid Resuscitation and balance  
 A = Analgesia  
 S = Sedation  
 T = Thromboembolic prophylaxis  
 H = Head-of-bed elevation  
 U = stress Ulcer prophylaxis, and  
 G = Glucose/glycemic control

## **CING-KUF: Diabetic ketoacidosis management**

C = Creatinine (check it)/ Catheterize  
 I = Insulin (5u/hour. Note: sliding scale no longer recommended in the UK)  
 N = Nasogastric tube (if patient comatose)  
 G = Glucose (once serum levels drop to 12)  
 K = K<sup>+</sup> (potassium)  
 U = Urea (check it)  
 F = Fluids (crystalloids)

## **GET SMASHED: Causes of acute pancreatitis**

G = Gallstones  
 E = Ethanol  
 T = Trauma  
 S = Steroids  
 M = Mumps  
 A = Autoimmune (PAN)  
 S = Scorpion bites  
 H = Hyperlipidemia  
 E = ERCP  
 D = Drugs (azathioprine, diuretics)

## **HEADS: Stroke risk factors**

H = Hypertension/ Hyperlipidemia  
 E = Elderly  
 A = Atrial fibrillation  
 D = Diabetes mellitus/ Drugs (cocaine)  
 S = Smoking/ Sex (male)

## **HEAD HEART VESSELS: Syncope causes, by system** **CNS causes include HEAD:**

H = Hypoxia/ Hypoglycemia  
 E = Epilepsy  
 A = Anxiety  
 D = Dysfunctional brain stem (basivertebral TIA)

## **Cardiac causes are HEART:**

H = Heart attack  
 E = Embolism (PE)  
 A = Aortic obstruction (IHSS, AS or myxoma)  
 R = Rhythm disturbance  
 T = ventricular Tachycardia

## **Vascular causes are VESSELS:**

V = Vasovagal  
 E = Ectopic (reminds one of hypovolemia)  
 S = Situational  
 S = Subclavian steal  
 E = ENT (glossopharyngeal neuralgia)  
 L = Low systemic vascular resistance  
 (Addison's, diabetic vascular neuropathy)  
 S = Sensitive carotid sinus

### **HEPATICS: Hepatic encephalopathy, precipitating factors**

H = Hemorrhage in GIT/ Hyperkalemia  
 E = Excess protein in diet  
 P = Paracentesis  
 A = Acidosis/ Anemia  
 T = Trauma  
 I = Infection  
 C = Colon surgery  
 S = Sedatives

### **HARD-UPS: Causes of Normal Anion Gap Metabolic Acidosis**

H = Hyperventilation (chronic)  
 A = Acetazolamide, Acids (e.g., hydrochloric), Addison's disease

R = Renal tubular acidosis  
 D = Diarrhea  
 U = Ureterosigmoidostomy  
 P = Pancreatic fistulas and drainage  
 S = Saline (in large amounts)  
 (hyperchloremic metabolic acidosis)

### **HOLT: Jugular venous pressure elevation causes**

H = Heart failure  
 O = Obstruction of vena cava  
 L = Lymphatic enlargement – supraclavicular  
 T = intra-Thoracic pressure increase

### **INFARCTIONS: Myocardial infarction treatment**

I = IV access  
 N = Narcotic analgesics (e.g., morphine, pethidine)  
 F = Facilities for defibrillation (DF)  
 A = Aspirin/ Anticoagulant (heparin)  
 R = Rest  
 C = Converting enzyme inhibitor  
 T = Thrombolysis  
 I = IV beta blocker  
 O = Oxygen

N = Nitrates  
 S = Stool Softeners

### **KUSSMAL: Causes of High AG Metabolic Acidosis**

K = dKA  
 U = Uremia  
 S = Salicylates poisoning  
 S = Sepsis  
 M = Methanol poisoning  
 A = Alcoholic ketoacidosis  
 L = Lactic acidosis

### **LEMON: Difficult laryngoscopy**

L = Look externally, e.g. short neck, large tongue, large teeth, etc  
 E = Evaluate 3-3-2  
 – 3 = adequacy of oral access  
 – 3 = to assess capacity of mandibular space to accommodate tongue  
 – 2 = distance of larynx to level of base of tongue  
 M = Mallampati scoring  
 O = Obstruction  
 N = Neck mobility

## LOAD: Rapid Sequence Intubation Premedications

L = Lidocaine  
O = Opioids  
A = Atropine  
D = Defasciculating dose of competitive NMB

## MANTRELS: Clinical Decision Rules for Appendicitis

M = Migration of pain RLQ  
A = Anorexia  
N = N/V  
T = Tenderness in RLQ  
R = Rebound pain  
E = Elevated temp  $\geq 37.3$  C  
L = Leukocytosis  $\geq 10$   
S = Shift of WBC to left

## METHODS Discharge Planning

M = Medication  
E = Environment and exercise  
T = Treatment  
H = Health Teachings  
O = Out Patient

D = Diet  
S = Spiritual Nursing

## MOANS: Difficult BVM

M = Mask seal not good, e.g. beard, facial deformity, etc  
O = Obesity (difficult ventilate), 3rd trimester pregnancy, or obstruction e.g. neck swelling, angioedema, hematomas, cancer, etc  
A = Age, elderly, loss of muscle tone to support the upper airway  
N = No teeth (no teach causing caved in face)  
S = Stiff lungs – upper airway obstruction – exacerbation of asthma, COPD, etc

## MEALSSS: Rapid Sequence Intubation Equipments

M = Mask – well fitting snugly  
E = ETT (appropriate size + 1 size above and 1 size below); for children, ETT size =  $(\text{age}/4) + 4$  or child's little finger (less accurate)  
A = Airway gadgets in case of difficult airway or failed airway, e.g. Oropharyngeal airways, surgical airways,

Quicktrach, cricothyrotomy set, LMAs  
L = Laryngoscope blade: Straight (in BM, "L"urus, therefore, Mi"L"er), "C"urved = Ma"C"intosh; good light source  
S = Syringe to test and inflate cuff balloon of ETT  
S = Stylet  
S = Suction catheter, Yankauer catheter

## MIDAS: Coma (conditions to exclude as cause)

M = Meningitis  
I = Intoxication  
D = Diabetes  
A = Air (respiratory failure)  
S = Subdural/ Subarachnoid hemorrhage

## OLDER SAAB: Pain history checklist

O = Onset  
L = Location  
D = Description (what does it feel like)  
E = Exacerbating factors  
R = Radiation  
S = Severity  
A = Associated symptoms

A = Alleviating factors

B = Before (ever experience this before)

## **O NAVEL: Endotracheal tube deliverable drugs**

O = Oxygen

N = Naloxone

A = Atropine

V = Ventolin (albuterol), Vasopressin

E = Epinephrine

L = Lidocaine

## **O SHIT: Management of acute severe asthma**

O = Oxygen (high dose: >60%)

S = Salbutamol (5mg via oxygen-driven nebuliser)

H = Hydrocortisone (or prednisolone)

I = Ipratropium bromide (if life threatening)

T = Theophylline (or preferably aminophylline-if life threatening)

## **PATCH MED: Pulseless electrical activity – causes**

P = Pulmonary embolus

A = Acidosis

T = Tension pneumothorax

C = Cardiac tamponade

H = Hypokalemia/ Hyperkalemia/ Hypoxia/ Hypothermia/ Hypovolemia

M = Myocardial infarction

E = Electrolyte derangements

D = Drugs

## **PIRATES: Atrial fibrillation causes**

P = Pulmonary: PE, COPD

I = Iatrogenic

R = Rheumatic heart: mitral regurgitation

A = Atherosclerotic: MI, CAD

T = Thyroid: hyperthyroid

E = Endocarditis

S = Sick sinus syndrome

## **PQRST: Mnemonic for a complete pain history**

P3 = Positional, palliating, and provoking factors

Q = Quality

R3 = Region, radiation, referral

S = Severity

T3 = Temporal factors (time and mode of onset, progression, previous episodes)

## **PULSE: MI signs and symptoms**

P = Persistent chest pains

U = Upset stomach

L = Lightheadedness

S = Shortness of breath

E = Excessive sweating

## **RATE: Hemolytic-Uremic Syndrome components**

R = Renal failure

A = Anemia (microangiopathic, hemolytic)

T = Thrombocytopenia

E = Encephalopathy (TTP)

## **RESS: Principles of management in toxicology**

R = Reduce absorption

E = Enhance elimination

S = Specific antidote

S = Supportive treatment

## **RN CHAMPS: Shock types**

R = Respiratory

N = Neurogenic

C = Cardiogenic

H = Hemorrhagic



A = Anaphylactic  
M = Metabolic  
P = Psychogenic  
S = Septic

## **RODS: Difficult extraglottic devices**

R = Restricted mouth opening  
O = Obstruction upper airway  
D = Disrupted or distorted upper airway  
S = Stiff lungs, spine of cervical

## **SAD PUCKER: Structures in retroperitoneal space**

S = Suprarenal glands (adrenals)  
A = Aorta/IVC  
D = Duodenum (2nd – 3rd, and 4th segments)  
P = Pancreas (tail is intraperitoneal)  
U = Ureters  
C = Colon (only the ascending and descending parts)  
K = Kidneys  
E = Esophagus  
R = Rectum

## **SAMPLE: Focused History in Emergency Conditions/Trauma**

S = signs and symptoms  
A = allergies  
M = medications  
P = pertinent past medical history  
L = last oral intake  
E = events leading up to.

## **Scared Lovers Try Positions That They Can't Handle: Carpal (Wrist) Bones**

S = Scaphoid  
L = Lunate  
T = Triquetrum  
P = Pisiform  
T = Trapezium  
T = Trapezoid  
C = Capitate  
H = Hamate

## **SHORT: Difficult cricothyrotomy**

S = previous Surgery  
H = Hematoma/swelling around neck  
O = Obesity

R = Radiation distortion  
T = Tumor

## **SIMPLE: Criteria to define simple febrile seizure**

S = Seizure of focal type  
I = Intracranial infection  
M = Multiple times a day  
P = Past history of afebrile seizure  
L = Last longer than 15 minutes  
E = Examination abnormalities

## **SINUS BRADICARDIA (sinus bradycardia): Sinus bradycardia aetiology**

S = Sleep  
I = Infections (myocarditis)  
N = Neap thyroid (hypothyroid)  
U = Unconsciousness (vasovagal syncope)  
S = Subnormal temperatures (hypothermia)  
B = Biliary obstruction  
R = Raised CO<sub>2</sub> (hypercapnia)  
A = Acidosis  
D = Deficient blood sugar (hypoglycemia)  
I = Imbalance of electrolytes

C = Cushing's reflex (raised ICP)  
 A = Aging  
 R = Rx (drugs, such as high-dose atropine)  
 D = Deep anaesthesia  
 I = Ischemic heart disease  
 A = Athletes

### **SITTT: Causes of hematuria**

S = Stone  
 I = Infection  
 T = Trauma  
 T = Tumor  
 T = Tuberculosis

### **TOM SCHREPFER: Predisposing Conditions for Pulmonary Embolism**

T = Trauma  
 O = Obesity  
 M = Malignancy  
 S = Surgery  
 C = Cardiac disease  
 H = Hospitalization  
 R = Rest (bed-bound)  
 E = Estrogen, pregnancy, post-partum  
 P = Past hx

F = Fracture  
 E = Elderly  
 R = Road trip

### **TV SPARC CUBE: Shock signs and symptoms**

T = Thirst  
 V = Vomiting  
 S = Sweating  
 P = Pulse weak  
 A = Anxious  
 R = Respirations shallow/rapid  
 C = Cool  
 C = Cyanotic  
 U = Unconscious  
 B = BP low  
 E = Eyes blank

### **USED CARP: Causes of Normal Anion Gap Metabolic Acidosis**

U = Ureteroenterostomy  
 S = Small bowel fistula  
 E = Extra chloride  
 D = Diarrhea  
 C = Carbonic anhydrase inhibitors  
 A = Adrenal insufficiency

R = Renal tubular acidosis  
 P = Pancreatic fistula

### **VOMITING: Vomiting, extra GI differential**

V = Vestibular disturbance/ Vagal (reflex pain)  
 O = Opiates  
 M = Migraine/ Metabolic (DKA, gastroparesis, hypercalcemia)  
 I = Infections  
 T = Toxicity (cytotoxic, digitalis toxicity)  
 I = Increased ICP, Ingested alcohol  
 N = Neurogenic, psychogenic  
 G = Gestation

**References and Further Reading, click [here](#)**

# Classifications and Scores

by Sarah Attwa and Marwan Galal

## Case 1

*A 20-year-old male presents to your ED with a 5 cm wound after he fell off his motorbike. On physical exam, the wound overlays a fractured left tibia but does not show extensive soft tissue damage nor any signs of periosteal stripping or vascular injury. Which antibiotic should you give to this patient?*

**Table 20.6** Gustilo-Anderson Classification

TYPE	DEFINITION
Type I	Open fracture, clean wound, wound <1cm in length
Type II	Open fracture, wound >1cm in length without extensive soft tissue damage, flaps, avulsions
Type III	Open fracture with extensive soft tissue laceration, damage, or loss or an open segmental fracture. This type also includes open fractures caused by farm injuries, fractures requiring vascular repair, or fractures that have been open for 8 hours prior to treatment.
Type III A	Type III fracture with adequate periosteal coverage of the fractured bone despite extensive soft tissue laceration or damage
Type III B	Type III fracture with extensive soft tissue loss and periosteal stripping and bone damage. Usually associated with massive contamination. It will often need further soft tissue coverage procedure (i.e. free or rotational flap).
Type III C	Type III fracture associated with arterial injury requiring repair, irrespective of degree of soft tissue injury

**Application:** Gustilo-Anderson classification for open wounds and antibiotic coverage

**Interpretation:** According to the above classification, each class should receive the following antibiotics:

Type I: 1st generation cephalosporin

Type II: 1st generation Cephalosporin +/- Gentamycin

Type III: 1st generation Cephalosporin + Gentamycin +/- Penicillin

**Hint:** In farm and war wounds, all 3 antibiotics must be given

**The answer** to the above clinical scenario: Type II, Cephazolin +/- Gentamycin

## Case 2

*A 7-year-old boy was brought by his mother to the ED after a heavy object fell on his right hand earlier the same day. On physical exam, there is bony tenderness, swelling, and erythema over his right middle finger PIP joint. Distal pulses are intact and no neurological deficit. You decided to send the patient for imaging. XR is shown below. What is the classification of this fracture?*

Image 20.1



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## Illustration 20.1 Salter-Harris Classification



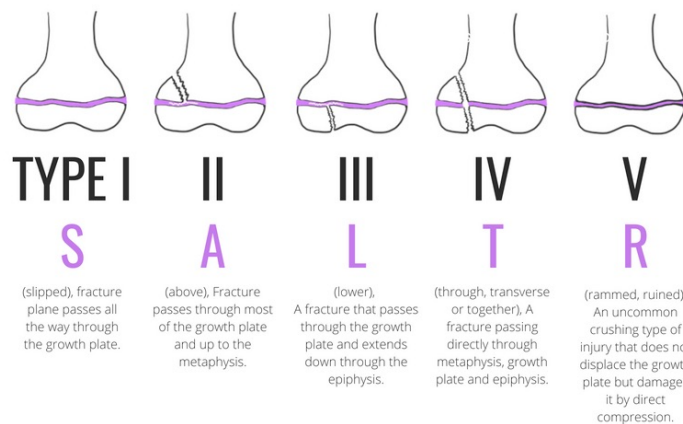
INTERNATIONAL  
EMERGENCY  
MEDICINE  
EDUCATION  
PROJECT

# Ortho Pearls

## SALTER - HARRIS CLASSIFICATION

Salter - Harris Fractures involve the physis and cartilaginous epiphyseal plate near the ends of the long bones in still growing children and adolescents. Damage to the growth plate during growth may destroy part or all of its ability to produce new bone, thus preventing elongation of the bone, which may lead to anatomical and functional deformities.

Salter-Harris fracture types can be memorized by the mnemonic SALTR.



Increase in Type of the fracture (I to V) increases the risk of complications.



**Application:** This a classification for long bone fractures involving epiphyseal growth plates.

### Hints:

Class II fractures are the most common injuries seen in the ED

Class V and I are the least commonly picked up

Class V carries the worst prognosis

**The answer** to the above clinical scenario: Class II

### Case 3

*An 85-year-old female was brought to the ED by her son with a 2-day history of fever and altered mental status. She is known to be diabetic and hypertensive. Her vitals are Temp 38.6 Celsius, BP 85/53, HR 110/min and RR is 26/min, and O2 saturation is 98% on room air. On examination, she is alert but confused, and the rest of her physical exam is unremarkable. Random glucose level is 8.5 mmol/L,*

*CXR is normal, CBC shows WBC of 3600 and urine dipstick is positive for nitrites and leukocytes. What is the next step in management?*

### SIRS (Systemic Immune Response Syndrome) Formula

1. Temp > 38 c or < 36 c
2. HR > 90/min
3. RR > 20/min or PaCO<sub>2</sub> < 32 mmHg
4. WBC > 12000 or < 4000

**Application:** Any patient with suspected systemic inflammatory response and can help guide critical decisions and interventions

**Interpretation:** SIRS is met when the patient has 2 or more criteria of the above

### Hints:

SIRS + source of infection = Sepsis (16% Mortality)

Sepsis + more than one organ's system dysfunction (e.g. Oliguria) = Severe Sepsis (20% Mortality)

Severe Sepsis + Hypotension (unresponsive to fluid resuscitation) = Septic Shock (69% Mortality)

**The answer** to the above clinical scenario: By applying the above criteria, this patient has SIRS + urinary tract infection; therefore, she is in sepsis. She needs adequate fluid resuscitation + Antibiotics, and if still hypotensive, she will be classified as septic shock and will require a higher level of care (e.g., Vasopressors) and close monitoring. Those patients should be admitted to the ICU.

## Case 4

*A 27-year-old female presented to the ED with severe abdominal pain for 1 day. No allergies or significant past medical history. Her vitals are:*

*Temp 37.6 Celsius, BP 100/55, HR 110/min, RR 20/min and O2 Saturation is 99% on room air. What level of care does this patient require?*

## Formula

SHOCK INDEX (SI) = HR / SBP

**Application:** It can be used to identify patients needing a higher level of care despite vital signs that may not appear strikingly abnormal. This index is a sensitive indicator of left ventricular dysfunction and can become elevated following a reduction in left ventricular stroke work.

## Interpretation:

Normal SI = 0.5 to 0.7

If SI > 0.9 was helpful to identify patients in the ED requiring admission and/or intensive care despite apparently stable vital signs

Persistent high SI has been associated with poor outcome

**The answer** to the above clinical scenario: By applying the above equation, ( $110/100 = 1.1$ ), this patient has a high shock index and requires a high level of care.

## Case 5

*A 72-year-old female presented with a fever, cough, and sputum for the last 4 days. She has a past medical history of DM and hypertension. Her vitals are: Temp 38.9 Celsius, HR 110/min, BP 100/45, RR 27/min, and O2 sat 92% on room air. On exam, she is alert and oriented, and chest auscultation reveals crackles over the right lower chest. The remainder of the physical exam*

*was normal. CXR reveals right lower lung lobe infiltrate. Labs showed mildly elevated white cell count with normal renal function and metabolic panel. How would you risk-stratify the severity of pneumonia in this patient? What would be her appropriate disposition?*

**Table 20.7** CURB-65

CATEGORY	CRITERIA	SCORE
C	Confusion	1
U	Urea > 7 mmol/L	1
R	Respiratory rate > 30	1
B	Systolic BP <90mmhg or Diastolic BP <60 mmHg	1
65 years	Age > 65 years	1
<b>CURB-65 score</b>	<b>30-day mortality</b>	<b>Management</b>
0-1	<5%	Home
<b>2</b>	<10%	Likely to need admission
3-5	15-30%	Admit, manage as severe

**Application:** Clinical scoring system used for risk stratification and guide management in all adult patients presenting with evidence of pneumonia

**The answer** to the above clinical scenario: By applying the above tool, this patient has a CURB-65 score of 2 (age + diastolic BP). This patient will likely need admission for further treatment.

## Case 6

*A 61-year-old female presents to the ED with leg swelling over the past 2 days. Her past medical history is positive for DM, hypertension, treatment for ovarian cancer 4 months ago. Her vital signs are Temp, 37.8 C, HR: 98, BP: 109/72, RR: 16, and O2 sat 98% on room air. On exam, she is alert, oriented and in acute distress. Lung auscultation is clear;*

*cardiac auscultation reveals normal S1-S2 with tachycardia, no lower limb edema or tenderness. Her left leg is swollen compared to right side. 12 lead ECG shows sinus rhythm with no abnormal findings. What is your next step in diagnosis?*

**Application:** Wells score is used to calculate pretest probability for all patients with clinically suspected DVT

## Interpretation:

0: low pretest probability

1-2: Moderate pretest probability

3 or more: High pretest probability

**Table 20.8** Wells Score for Deep Vein Thrombosis

CRITERIA	SCORE
Active cancer(treatment ongoing or within previous 6 months or palliative treatment)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden for 3 days or more or major surgery within the previous 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling > 3cm compared to asymptomatic leg (measuring 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Non varicose collateral superficial veins	1
Previously documented DVT	1
Alternative diagnosis at least as likely as DVT	1

## Case 7

*A 54-year-old male with a past medical history of peripheral vascular disease comes in with on/off palpitations and lightheadedness for the past 2 weeks. His vital signs are normal. On exam, he is alert, oriented and chest is clear to auscultation and heart sounds are irregularly irregular. The remainder of his physical exam is unremarkable. His ECG shows Atrial Fibrillation with HR of 96 beats/min. What is this patient's risk for*

*developing stroke? What is the recommended therapy for him?*

**Table 20.9** CHADS2 Score for Atrial Fibrillation

CATEGORY	CRITERIA	POINT VALUE
C	Congestive heart failure	1
H	Hypertension (>140/90 mmHg)	1
A	Age > 75 years	1
D	Diabetes Mellitus	1
S2	Prior Stroke or TIA	2

**Application:** Clinical prediction rule for assessing the risk of stroke in patients with non-rheumatic Atrial Fibrillation and is used to determine if treatment is required with anticoagulation therapy or antiplatelet therapy or not.

**Table 20.10** CHADS2 Interpretation

SCORE	RISK	ANTICOAGULATION THERAPY	RECOMMENDATIONS
0	Low	No therapy OR Aspirin (ASA)	No therapy, if patient prefers give ASA
1	Moderate	Oral anticoagulant OR ASA	Oral anticoagulant, alternatives are ASA with Clopidogrel or ASA alone
2 or greater	High	Oral anticoagulant	Oral anticoagulant, alternatives are ASA with Clopidogrel or ASA alone



## Special considerations

- In low-risk patients, female sex OR patients with vascular disease, ASA is recommended
- In low-risk patients, female sex AND vascular disease, oral anticoagulant is preferred
- In low-risk patients, if age > 65 years, oral anticoagulant is preferred

**The answer** to the above clinical scenario: Applying the above score, the patient is at low risk for stroke (Score of 0), and the recommended therapy for him, given his peripheral vascular disease is ASA.

## Case 8

*A 70-year-old male with known ischemic heart disease and permanent pacemaker presents to the ED with chest pain for 2 hours. His initial vitals are stable and below is his*

## ECG. How do you interpret this ECG?

### Sgarbossa Criteria

- Concordant ST elevation  $\geq 1$  mm = 5 points
- ST depression  $\geq 1$  mm in V1-V3 = 3 points
- Discordant ST elevation  $\geq 5$  mm = 2 points

**Application:** Used in cases of left bundle branch block (LBBB) and suspicion of acute myocardial infarction (AMI)

**Interpretation:** At score-sum of 3 or greater, these criteria have specificity of 90% for detecting AMI

**Hints:** These criteria can also be applied to Pacemaker rhythm

**The answer** to the above clinical scenario: This ECG is showing a paced rhythm, and you can clearly see the pacemaker spikes. By applying the above criteria, this patient has Concordant ST

depression in V2 and V3 > 1mm, which gives him a Sgarbossa score of 3. This means this patient has Acute MI.

## Case 9

*A 13-year-old boy comes in complaining of right lower quadrant abdominal pain for the past 2 days, associated with nausea, vomiting, and loss of appetite. His vitals are temperature 38.1, BP 110/77, HR 100, RR 18, and oxygen saturation 99% on RA. On physical exam, he has right iliac fossa tenderness with rebound. His initial labs are significant for leukocytosis with neutrophilic left shift. After pain relief, what is the next best step in management?*

**Table 20.11** Alvarado Score for Appendicitis

CATEGORY	CRITERIA	POINT VALUE
Symptoms	Migratory right iliac fossa pain	1
	Nausea/vomiting	1
	Anorexia	1
Signs	Tenderness in right iliac fossa	2
	Rebound tenderness in right iliac fossa	1
	Fever	1
Lab findings	Leukocytosis	2
	Neutrophil left shift	2

**Application:** Used in all cases of clinically suspected acute appendicitis

**Table 20.12** Interpretation of Alvarado Score

SCORE	SIGNIFICANCE
1-4	Unlikely
5-6	Possible
7-8	Acute appendicitis present
9-10	Definite acute appendicitis requiring surgery

**The answer** to the above clinical scenario: Using the above scoring system, the patient has a score of 9, and therefore,

definitely has acute appendicitis requiring surgery. He should be urgently referred to the surgical team.

### Case 10 and 11

*A 70-year-old male was brought to the ED by his son after he collapsed at home 2 hours ago. He has history of fever and URI symptoms for the past 3 days. On exam, patient stuporous, and opens his eye to a verbal prompt, moaning and withdraws from painful stimuli. What’s his GCS score?*

*A 18-year-old male involved in a motor vehicle collision was brought in by EMS with apparent facial and head injuries. On exam, with pinching his chest, he does not open his eyes nor makes any sounds but flexes both arms inwards. What’s his GCS score?*

**Table 20.13** Glasgow Coma Scale

CHOOSE THE BEST RESPONSE OF PATIENT
<b>EYE OPENING</b>
4: Spontaneously
3: To verbal command
2: To pain
1: No response
<b>BEST VERBAL RESPONSE</b>
5: Oriented and converses
4: Disoriented and converses
3: Inappropriate words; cries
2: Incomprehensible sounds
1: No response
<b>BEST MOTOR RESPONSE</b>
6: Obeys command
5: Localizes pain
4: Flexion withdrawal
3: Flexion abnormal (decorticate)
2: Extension (decerebrate)
1: No response

*Glasgow Coma Score (GCS) (Modified from Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. The Lancet, 304(7872), 81-84.) - Please read this article to get more insight regarding GCS.*

**Application:** Part of neurological examination for any patient (e.g., trauma, altered mental status, intoxication, etc.)

**Interpretation:** Useful objective tool to assess and quantify neurological function of patients in ED to help guide critical decisions and interventions (e.g., Intubation to protect the airway )

**Hints:** Patients with a score of 8 or below due to irreversible causes need airway protection via intubation

**Special consideration:** Modified GCS score for the pediatric population

**The answer** to the above clinical scenarios:

Case 10 – GCS score of 9

Case 11 – GCS score of 5 (this patient needs airway protection)

**References and Further Reading**, click [here](#)